April 1992
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The β-Agonist Dilemma (editorial)—B Burrows, MD Lebowitz. N Engl J Med 1992;326:560-561. (Relates to "Use of β-Agonists and the Risk of Death..." by WO Spritzer et al abstracted below.)

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The Use of β-Agonists and the Risk of Death and Near Death From Asthma—WO Spitzer, S Suissa, P Ernst, RI Horwitz, B Habbick, D Cockcroft, JF Boivin, M McNutt, AS Buist, AS Rebuck. N Engl J Med 1992;326:501.

BACKGROUND: Morbidity and mortality from asthma appear to be increasing, and it has been suggested that medications used to treat asthma are contributing to this trend. We investigated a possible association between death or near death from asthma and the regular use of β-agonist bronchodilators. METHODS: Using linked health insurance data bases from Saskatchewan, Canada, we conducted a matched case-control study of subjects drawn from a cohort of 12,301 patients for whom asthma medications had been prescribed between 1978 and 1987. We matched 129 case patients who had fatal or near-fatal asthma with 655 controls (who had received medications for asthma but had not had fatal or near-fatal events) with respect to region of residence, age, receipt of social assistance, and previous hospitalization for asthma. RESULTS: The use of β-agonists administered by a metered-dose inhaler was associated with an increased risk of death from asthma (odds ratio, 2.6 per canister per month; 95% confidence interval, 1.7 to 3.9) and of death or near death from asthma, considered together (odds ratio, 1.9; 95% confidence interval, 1.6 to 2.4). For death from asthma, use of the β-agonist fenoterol was associated with an odds ratio of 5.4 per canister, as compared with 2.4 for the β-agonist albuterol. On a microgram-equivalent basis, the odds ratio for this outcome with fenoterol was 2.3, as compared with 2.4 with albuterol. CONCLUSIONS: An increased risk of death or near death from asthma was associated with the regular use of inhaled β-agonist bronchodilators, especially fenoterol. Regardless of whether β-agonists are directly responsible for these adverse effects or are simply a marker for more severe asthma, heavy use of these agents should alert clinicians that it is necessary to re-evaluate the patient's condition.

Inadvertent Loss of Bronchoscopy Instruments in the Tracheobronchial Tree—JM Roach, G Rippe, TA Dillard. Chest 1992; 101:568.

Equipment malfunction is a rare complication of flexible fiberoptic bronchoscopy. We report an unusual example of equipment failure resulting in the inadvertent introduction of a foreign body (the tip of a cleaning brush) into the bronchial tree of a patient during FFB. In addition, we review several other cases in which problems have been caused by malfunction of accessory equipment. Careful inspection of all cleaning and biopsy utensils should reduce the chance of iatrogenically introducing a foreign body from the bronchoscope channel into the patient's airway. We suggest that biopsy forceps are the best retrieval instrument to use when the foreign body involved is a brush or a piece of wire.

Infected Radial Artery Pseudoaneurysms Occurring after Percutaneous Cannulation—PS Falk, PE Scuderi, RJ Sherertz, SM Motsinger. Chest 1992;101:490.

During a 10-month period in 1988 at our institution, we identified 3 infected radial artery pseudoaneurysms (RAPAs) associated with arterial lines. A retrospective chart review to 1983 identified 3 additional cases, all

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occurring in 1986. In the 6-year period of 1983 through 1988, during which approximately 12,500 radial artery catheters were placed, the incidence of RAPA formation was 6/12,500 (0.048%). Five of the six cases were associated with *Staphylococcus aureus* infection. The duration of radial artery cannulation was significantly longer in patients who developed a pseudoaneurysm (12.5 days) than in those patients who did not suffer this complication (4.3 days). Patients in whom infected RAPAs occurred also tended to be older (mean 71.6 y) than the average age (54 y) for all patients admitted to the intensive care unit (ICU). They also tended to have long stays in the ICU prior to development of RAPA, the shortest stay being 11 days and the average being 51 days. Risk factors for the development of this complication may include advanced age, longer duration of catheterization and hospitalization, and infection with *S aureus*.

Exercise Testing in the Evaluation of Patients at High Risk for Complications from Lung Resection—RC Morice, EJ Peters, MB Ryan, JB Putnam, MK Ali, JA Roth. Chest 1992;101:356.

Exercise testing was performed on 37 patients with resectable lung lesions who were deemed inoperable because of any of the following risk factors: (1) $FEV_1 \leq 40\%$ of predicted; (2) radionuclide calculated postlobectomy $FEV_1 \leq 33\%$ of predicted; or (3) arterial $P_{CO_2} \geq 45$ torr. The patients who reached a peak level of oxygen consumption during exercise ($\dot{V}_{O_2Peak}$) of ≥ 15 mL · min · kg^{-1} were offered surgical treatment. Patients with a $\dot{V}_{O_2Peak}$ of < 15 mL · min · kg^{-1} were referred for nonsurgical management and excluded from the study. Eight patients underwent lung resection. Their pulmonary function revealed a severe obstructive lung defect with a group mean predicted FEV_1 of 40 (6) %, an FEV_1/FVC ratio of 47 (10), a radionuclide calculated postlobectomy FEV_1 of 31 (4)%, and a mean arterial P_{CO_2} of 44 (6) torr. No relationship was found between each patient's exercise performance and spirometric function. Six of the patients had an uncomplicated postoperative course. Two patients had complications but no patient died as a result of surgery or postoperative complications. All patients were discharged from the hospital within 22 days (mean = 9.8 days). We conclude that exercise testing is a useful complement to conventional cardiopulmonary evaluation used in selecting patients for lung resection.

Adult Respiratory Distress Syndrome: Sequence and Importance of Development of Multiple Organ Failure—RC Bone, R Balk, G Slotman, R Maunder, H Silverman, TM Hyers, MD Kerstein, and Prostaglandin E_1 Study Group. Chest 1992;101:320.

STUDY OBJECTIVE: To determine the epidemiology of multiple organ failure (MOF) in patients with the adult respiratory distress syndrome. PATIENTS: We followed up 50 patients with serial determinations of respiratory and nonrespiratory organ function for 7 days after diagnosis DESIGN: Data were stratified between patients who died and those who survived (defined as hospital discharge). MEASUREMENTS & RESULTS: Values that did not differ at any time between the two groups of patients included oxygen availability, oxygen consumption, oxygen extraction, P_{aCO_2}, respiratory rate, heart rate, systolic blood pressure, cardiac output, stroke index, systemic vascular resistance, and temperature. Patients who died had greater defects in oxygenation (from Day 1 through Day 7). They also exhibited decreased arterial oxygen content (from Day 1 to Day 4), decreased mixed venous oxygen content (Day 1) increased peak inspiratory pressure (present on Day 2, persisted to Day 5, reappeared on Day 7), decreased diastolic blood pressure (seen on Days 1 through 3, reappeared on Day 7), and increased mean pulmonary artery pressure (seen on Days 2 and 3). Nonsurvivors also exhibited greater degrees of thrombocytopenia (from Day 1 to Day 4). Decreases in pH (seen on Day 1, reappeared from Days 4 to 7), abnormalities in liver function (seen only on Day 1), and increases in serum creatinine levels (appeared on Day 7) were also observed. CONCLUSIONS: Multiorgan dysfunction (MOD) was frequently observed in both groups of patients. Alterations in organ function and the pattern of abnormalities were often subtle and would not be

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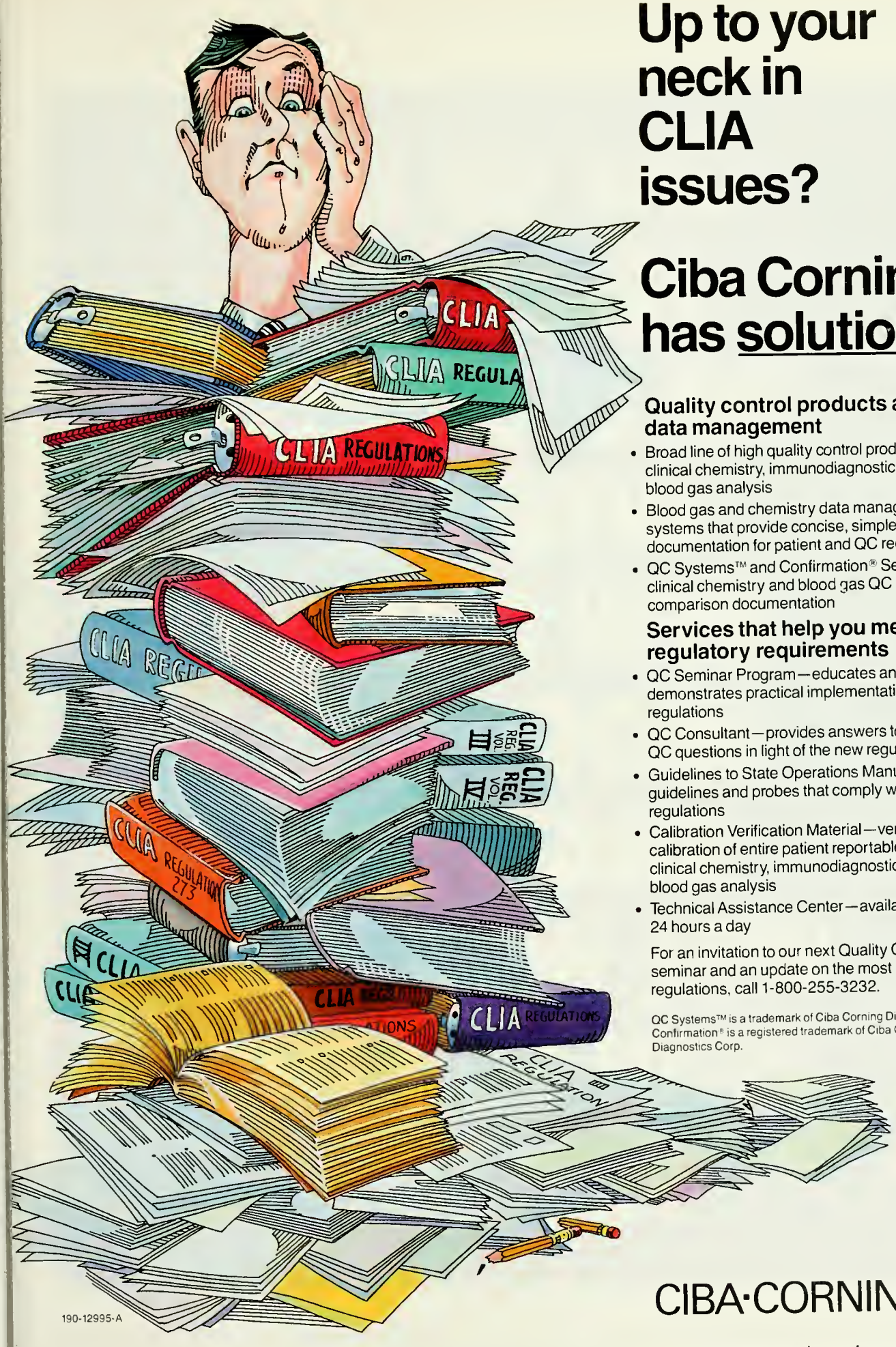

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The unit aspirates 90µL or 70µL samples directly from all common containers without the need for external adapters.

So with the BG3™ analyzer, when demands grow, and your needs grow, your capabilities grow with you.

But there's a lot more to the BG3™ analyzer than just upgradeability.

RELIABILITY.

IL's unique features of sequential sample introduction, full sample visibility to detect microbubbles and clots and short path design incorporating reverse flush fluidics eliminates bacterial contamination.

The BG3™ analyzer measures pH, pCO_2 and pO_2 and calculates up to 11 other parameters. It also continuously records time, date, barometric pressure and temperature.

MAINTENANCE.

With our disposable membrane caps, you can change membranes on all electrodes in minutes.

REGULATORY REQUIREMENTS (CLIA).

The system's continuous calibration feature monitors and updates electrode calibration every second, so it is always ready to accurately analyze a sample. It also performs a one-point calibration every 20 minutes and a two-point calibration at user-selected intervals.

With its on-board data-management system, the BG3 analyzer can store one year of quality control reports and over 6,000 complete patient reports.

DATA MANAGEMENT.

With the on-board data-management system, the BG3™ analyzer automatically maintains its own patient and quality control records. Patient trends and Levey Jennings charts are available at your fingertips. The system is so advanced, it even features a built-in maintenance calender that tells you when maintenance is needed. The BG3™ analyzer also interfaces with the IL IMPACT™ Blood Gas Data Management System allowing you to network other analyzers to meet all regulatory requirements.

Contact your IL representative about the new BG3™ Blood Gas Analyzer, and learn how you can meet your needs today and plan for your expanding needs in the future.

1991 ZENITH AWARD

Instrumentation Laboratory

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characterized as significant organ dysfunction by most available organ scoring systems. Adult respiratory distress syndrome is a manifestation of systemic disease produced by widespread increases in endothelial permeability; lung dysfunction dominates the early clinical course. When respiratory function is supported, it becomes evident that alterations occur in other organs. Multiorgan failure is really a misnomer; the term emphasizes end-stage changes. Multiorgan dysfunction is common and often resolves without progressing to MOF. Alternatively, MOD can progress to MOF.

Disposable Jet Nebulizers: How Reliable Are They?—GF Alvine, P Rodgers, KM Fitzsimmons, RC Ahrens. Chest 1992;101:316.

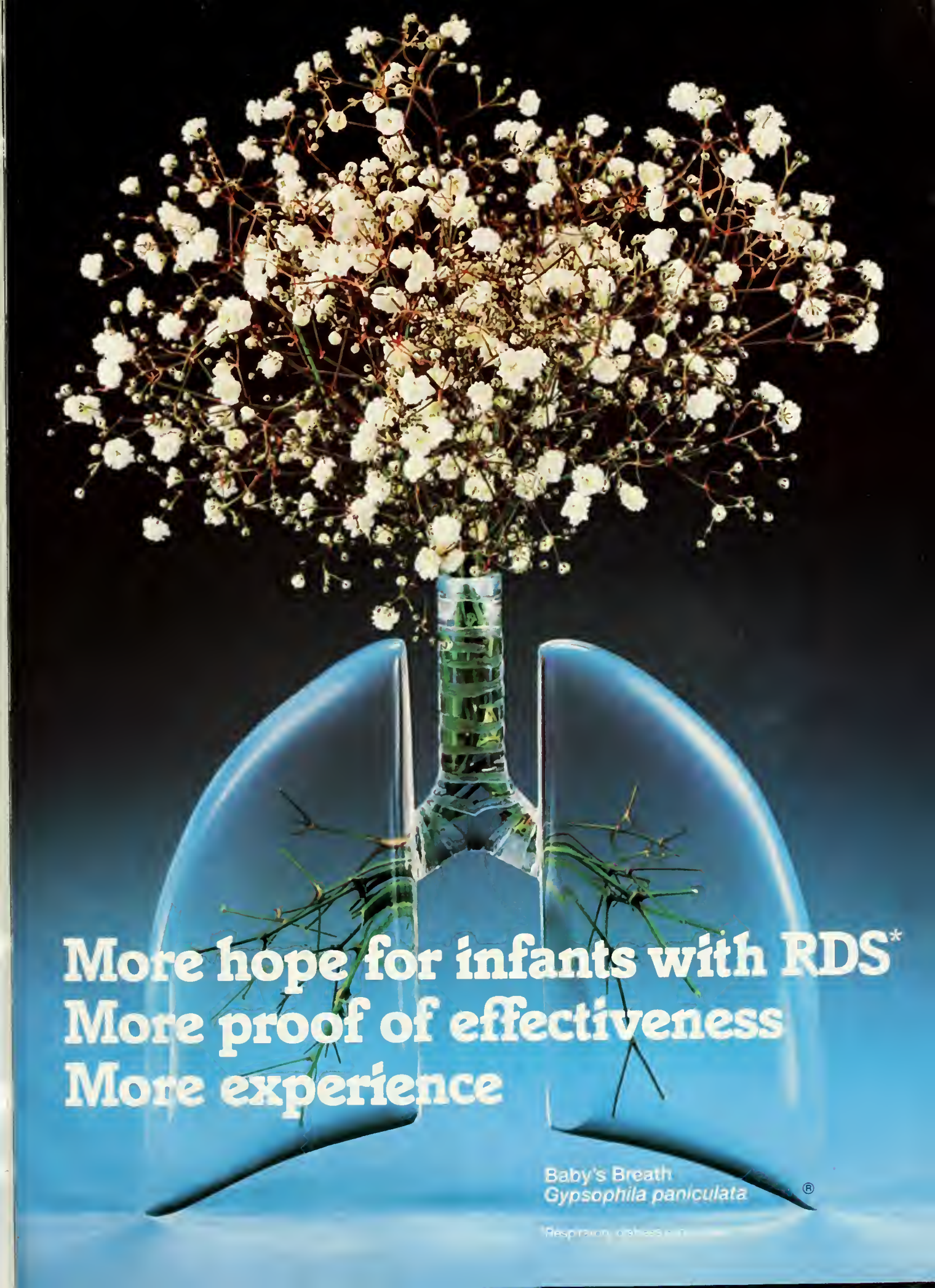
We studied the frequency of malfunction, variability in rate of nebulization, and effect of this variability on aerosol particle size of 8 disposable jet nebulizer models produced by 6 manufacturers. Four of eight models showed visual signs of malfunction, including spraying of large, individually visible droplets, leaking of nebulizer solution, and air leaks that completely prevented nebulization. Variability of nebulization rate within specific models ranged from 57 to 129%. The model with the largest variability of nebulization rate was also associated with an unacceptably large variability in particle size. In contrast, two models with smaller variability in nebulization rate had greater consistency of particle size. These results indicate poor quality control by some manufacturers in the disposable nebulizer industry. The data suggest that purchasing agents should consider reliability as well as cost before selecting a specific nebulizer model and that their evaluation should include physical testing of multiple units of each model under consideration.

Relative Amount of Albuterol Delivered to Lung Receptors from a Metered-Dose Inhaler and Nebulizer Solution: Bioassay by Histamine Bronchoprovocation—KV Blake, M Hoppe, E Harman, L Hendeles. Chest 1992;101:309.

The results of previous studies comparing bronchodilatation from beta agonists administered by metered-dose inhaler (MDI) and nebulizer solution have been conflicting. We therefore evaluated a range of albuterol doses administered by these on two methods, using histamine bronchoprovocation as a bioassay for the amount of drug reaching the β_2 receptors in the lung. Twelve stable asthmatic volunteers received in a double-blind, randomized, crossover design on different days, placebo or 1, 2, 4, or 6 puffs from an MDI attached to an InspirEase device (90 μg per puff) or 0.625, 1.25, 2.5, or 5.0 mg of solution delivered in 2 mL of buffered saline through a Hudson Updraft II nebulizer. The histamine concentration required to decrease FEV_1 by 20% (PC_{20}) was measured 1 h before and 30 min after administration of each treatment and expressed as the increase in PC_{20} from baseline. The dose-response curves for change in PC_{20} indicated that the higher doses of the nebulizer solution delivered more drug to β_2 receptors in the lung than the lower doses from the MDI. For example, the geometric mean (SD) increase in PC_{20} was 1.1 (1.6) after placebo, 7.5 (2.7) after 2 puffs from the MDI, and 20.0 (2.1) after 2.5 mg of nebulizer solution ($p < 0.05$). Using this bioassay method and administration technique, we estimated that 10 puffs from the MDI (0.9 mg) would deliver approximately the same amount of albuterol to lung receptors as 2.5 mg of the nebulizer solution. Taking into account previously published reports and the results of the present study, we conclude that differences in dose, administration technique, nebulizer system efficiency, and severity of airway obstruction can alter the amount of drug reaching the β_2 receptors in the lungs and, thus, the clinical response.

Substitution of Metered-Dose Inhalers for Hand-Held Nebulizers: Success and Cost Savings in a Large, Acute-Care Hospital—DL Bowton, WM Goldsmith, EF Haponik. Chest 1992;101:305.

Administration of β-agonist bronchodilators by metered-dose inhalers (MDI) is as effective as administration by hand-held nebulizer (NEB). Recent studies have suggested that MDI therapy is less costly to administer and that routine substitution of MDI for NEB would result in considerable savings to patients and to hospitals. To our knowledge, the actual extent to which MDI therapy would replace NEB therapy or the cost savings realized has not been reported previously. We examined the success and impact on hospital costs of the routine substitution of MDI for NEB therapy in a large tertiary-care hospital. Following introduction of this strategy, more than 60% of all aerosol therapy was actually given as MDI. The mean amount of time spent by therapists to provide aerosol therapy was significantly reduced by MDI substitution, falling from 1,576 (131) h/mo, to 992 (116) h/mo ($p < 0.002$). The total cost to deliver aerosol therapy fell from $27,600 ($2,277)/mo to $20,618 ($2,086)/mo ($p = 0.008$). Potential cost savings of $83,000 annually were achieved by the hospital, and charges to patients were lowered by approximately $300,000 per year. Routine substitution of MDI therapy


More hope for infants with RDS*
More proof of effectiveness
More experience
Baby's Breath
Gypsophila paniculata
®

The proven efficacy and impressive safety profile of the first protein-free, synthetic lung surfactant

Exosurf® NEONATAL™
(Colfosceril Palmitate, Cetyl Alcohol, Tyloxapol) For Intratracheal Suspension/10-mL vial

Controlled clinical trials with EXOSURF Neonatal showed "dramatic reductions" in morbidity and mortality of infants with RDS. Subsequent use under a year-long Treatment IND confirmed its efficacy and impressive safety profile. Since release for marketing in August 1990, widespread use in hospitals across the United States has further established its value in the treatment of RDS. The weight of clinical experience is in favor of EXOSURF Neonatal.

Dramatic reductions in neonatal morbidity and mortality reported in clinical trials[1-5]

Improvement in clinical outcome after EXOSURF Neonatal has been significant in infants at risk of developing RDS as well as those with established RDS. Prophylactic as well as rescue treatment with EXOSURF Neonatal has dramatically reduced morbidity and mortality in infants weighing greater than 700 grams.

SIGNIFICANT REDUCTIONS IN OVERALL MORTALITY FROM ANY CAUSE IN MIDDLE-SIZE AND LARGE INFANTS[2-5]
(Percent reductions with EXOSURF)

	Prophylactic treatment		Rescue treatment	
Age	*Single Dose*[4] 700-1100 grams (N=446)	*One vs Three Doses*[5] 700-1100 grams (N=716)	*Two Dose*[4] 700-1350 grams (N=419)	*Two Dose* 1250 grams and above (N=1232)
≤10 Days	—	—	59%[5]	35%
≤28 Days	40%*	44%[†]	52%[‡]	39%
≤1 Year	44%[†]	41%[†]	46%[‡]	31%

*P=.022 †P=0.002 ‡P<0.05. §P<0.001 ¶P=0.039. N=Number of infants enrolled in the clinical trials.

A single prophylactic dose of EXOSURF Neonatal reduced 1-year mortality by 44%. Two additional prophylactic doses provided an additional 41% reduction in 1-year mortality.[3]

SIGNIFICANT REDUCTIONS IN MORTALITY FROM RDS IN MIDDLE-SIZE AND LARGE INFANTS[2,4,5]
(Percent reductions with EXOSURF)

	Prophylactic treatment	Rescue treatment	
	Single Dose[4] 700-1100 grams (N=446)	*Two Dose*[4] 700-1350 grams (N=419)	*Two Dose* 1250 grams and above (N=1232)
Reduction in death from RDS	58%*	66%[†]	63%[‡]

*P=0.011 †P=0.002 ‡P=0.009 N=Number of infants enrolled in the clinical trials

SIGNIFICANT REDUCTION IN CARDIOPULMONARY DESTRUCTION TO DAY 28 FOLLOWING TWO-DOSE RESCUE TREATMENT[4,5]

(Percent reductions with EXOSURF)

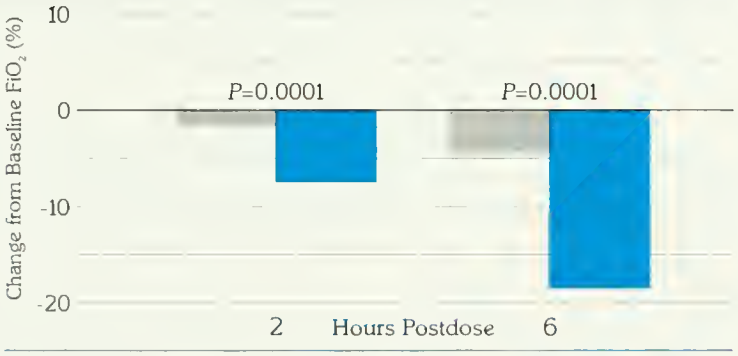
Birth weight	Reduction in death at 28 days or survival with broncho-pulmonary dysplasia (BPD)
700-1350[4] (N=419)	34%*
1250 and above[5] (N=1232)	43%*

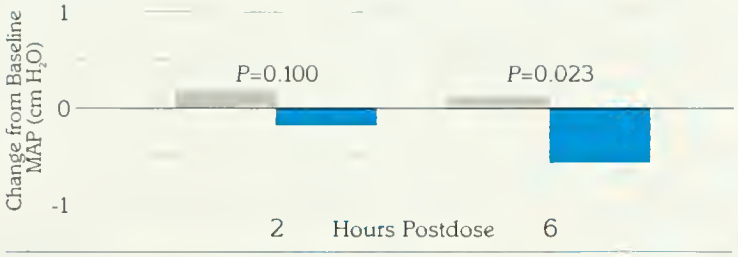
*P=0.002 N=Number of infants enrolled in the clinical trials

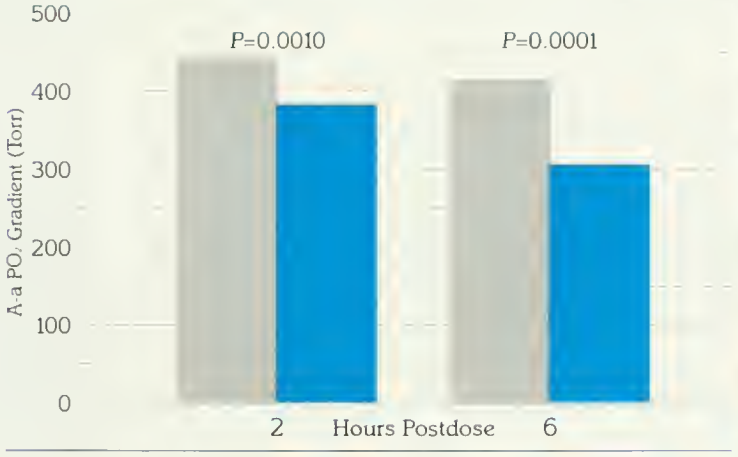
Rapid onset of action documented in rescue use[4]

Improvements in mean FiO_2 and mean alveolar-arterial PO_2 (A-a) gradient were present by 2 hours after dosing in middle-size babies (700-1350 grams). Improvements in mean airway pressures began sometime between 2 and 6 hours in middle-size babies. These improvements persisted for at least 7 days.

SIGNIFICANT IMPROVEMENTS IN SUPPLEMENTAL OXYGEN NEEDS AND VENTILATORY REQUIREMENTS SEEN IN MIDDLE-SIZE BABIES[4]







Air

EXOSURF Neonatal

All charts from Long[4]

Please see brief summary of full prescribing information on last pages of this advertisement.

Efficacy and impressive safety profile of EXOSURF® Neonatal™ confirmed in continued widespread use[6]

In North American controlled clinical trials, more than 2600 premature infants received EXOSURF Neonatal. Under the year-long Treatment IND, over 11,400 infants received EXOSURF Neonatal. In the six months following its release for marketing, EXOSURF Neonatal has been given to 10,000 infants in more than 750 hospitals.

There are no known infectious or immunologic risks associated with EXOSURF Neonatal use. In controlled clinical trials, adverse events were comparable to those of placebo, with the exception of apnea and pulmonary bleeding. Infants receiving EXOSURF Neonatal required less ventilatory support, possibly contributing to an increased incidence of apnea. Pulmonary bleeding occurred in 1% of control infants and 2% of treated infants in controlled trials. In the treatment IND, pulmonary bleeding was reported in 4% and mucous plugging at a rate of 3/1000. Pulmonary bleeding appears to be preventable with early diagnosis and appropriate treatment of patent ductus arteriosus.

One-year follow-up evaluated developmental outcomes[7]

Double-blind 1-year follow-up of more than 1450 infants enrolled in randomized trials showed that mental and motor scores appeared to be higher in tiny infants (<750 grams) as well as middle-size infants (750-1249 grams) who received Exosurf Neonatal.

Economic data analysis showed cost savings[8-10]

Three separate studies evaluated the economic impact of a single prophylactic dose of EXOSURF Neonatal, two-dose rescue treatment in 700- to 1350-gram infants, and two-dose rescue treatment during the neonatal period in infants weighing over 1350 grams. Results indicate that both prophylactic treatment and rescue treatment are cost-effective. Mean hospital charges were $6451 less for large infants receiving two-dose rescue treatment versus air in the first 28 days of life.[10]

As easy to use as it is effective

- **Easy to store and use** EXOSURF Neonatal may be stored at room temperature. Reconstituted suspension may be maintained refrigerated or at room temperature for up to 12 hours. Key items needed for administration are supplied in one kit.
- **Easy to administer** Each EXOSURF Neonatal dose is administered in two 2.5-mL/kg half-doses without interrupting mechanical ventilation.
- **Easy on infant** To assist the distribution of EXOSURF Neonatal in the lungs, the infant is simply turned from midline position to the right after the first half-dose, and from midline position to the left after the second half-dose.

References: 1. Bose C, Corbet A, Bose G, et al. Improved outcome at 28 days of age for very low birth weight infants treated with a single dose of a synthetic surfactant. *J Pediatr* 1990;117:947-953. **2.** Corbet A, Bucciarelli R, Goldman S, et al. Decreased mortality in small premature infants treated at birth with a single dose of synthetic surfactant: a multi-center controlled trial. *J Pediatr* 1991;118:277-284. **3.** Gerdes J, Cook L, Beaumont E, Corbet A, Long W. American EXOSURF Pediatric Groups I and II. Effects of three vs. one prophylactic doses of EXOSURF Neonatal in 700- to 1100-gram neonates. *Pediatr Res* 1991;29(no. 4, pt 2):214A. Abstract. **4.** Long W, Thompson T, Sundell H, et al. Effects of two rescue doses of a synthetic surfactant on mortality rate and survival without bronchopulmonary dysplasia in 700- to 1350-gram infants with respiratory distress syndrome. *J Pediatr* 1991;118:595-605. **5.** U.S. and Canadian EXOSURF Pediatric Study Groups. Effects of two rescue doses of EXOSURF® Pediatric in 1232 infants ≥1250 grams. *Pediatr Res* 1990;27(no. 4, pt 2):320A. Abstract. **6.** Data on file, Burroughs Wellcome Co., 1991. **7.** Walter D, McGuinness G, Bose C, et al. Double-blind one year follow-up in 1450 infants randomized to EXOSURF® Neonatal or air. *Pediatr Res* 1991;29(no. 4, pt 2):270A. Abstract. **8.** Sell M, Corbet A, Gong A, et al. Economic impact of a single prophylactic dose of EXOSURF® Neonatal in 700- to 1100-gram infants. *Pediatr Res* 1991;29(no. 4, pt 2):265A. Abstract. **9.** Mammel M, Mullett M, Derleth D, et al. Economic impact of two rescue doses of EXOSURF® Neonatal in 700-1350 gram infants. *Pediatr Res*. 1991;29(no. 4, pt 2):260A. Abstract. **10.** Schumacher R, Burchfield D, Vaughan R, et al. Economic impact of two rescue doses of EXOSURF® Neonatal in >1350 gram infants. *Pediatr Res*. 1991;29(no. 4, pt 2):264A. Abstract.

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Wellcome

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EX Y01228

EXOSURF® Neonatal™
(COLFOSCERIL PALMITATE, CETYL ALCOHOL, TYLOXAPOL)
For Intratracheal Suspension / 10 mL vials

PLEASE CONSULT FULL PRODUCT INFORMATION BEFORE PRESCRIBING

INDICATIONS AND USAGE Exosurf Neonatal is indicated for

1 **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS)

2 **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity and

3 **Rescue** treatment of infants who have developed RDS

CONTRAINDICATIONS There are no known contraindications to treatment with Exosurf Neonatal

WARNINGS. Intratracheal Administration Only Exosurf Neonatal should be administered only by instillation into the trachea **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants. Instillation of Exosurf Neonatal should be performed **only** by trained medical personnel experienced in airway and clinical management of unstable premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal. **Acute Effects.** Exosurf Neonatal can rapidly affect oxygenation and lung compliance. **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak. **Hyperoxia.** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment. Failure to reduce FiO_2 in such instances can result in hyperoxia. **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow. **Pulmonary Hemorrhage.** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, pulmonary hemorrhage was reported for 1% (14/1420) of infants in the placebo group and 2% (27/1411) of infants in the Exosurf Neonatal group. Fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group. Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. In more than 7700 infants in the open, uncontrolled study, pulmonary hemorrhage was reported in 4%, but fatal pulmonary hemorrhage was reported rarely (0.4%). In the controlled clinical studies, Exosurf Neonatal treated infants who received steroids more than 24 hours prior to delivery or indomethacin postnatally had a lower rate of pulmonary hemorrhage than other Exosurf Neonatal treated infants. Attention should be paid to early and aggressive diagnosis and treatment (unless contraindicated) of patent ductus arteriosus during the first 2 days of life (while the ductus arteriosus is often clinically silent). Other potentially protective measures include attempting to decrease FiO_2 preferentially over ventilator pressures during the first 24 to 48 hours after dosing, and attempting to decrease PEEP minimally for at least 48 hours after dosing. **Mucous Plugs:** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately.

PRECAUTIONS:

General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies or who were suspected of having congenital infection, were excluded from entry. However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered. The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection. **Prophylactic Treatment–Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS). Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups (see Table 1 in full product information). Data on multiple doses in infants in this weight class are not yet available. Accordingly, clinicians should carefully evaluate the potential risks and benefits of Exosurf Neonatal administration in these infants. **Rescue Treatment–Number of Doses.** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 µg/plate was not mutagenic in the Ames Salmonella assay. Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal. The effects of Exosurf Neonatal on fertility have not been studied.

ADVERSE REACTIONS

General: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died.

In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made. In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased. A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. Tables 3 and 4 summarize the results of the major safety evaluations from the controlled clinical studies.

Table 3 Safety Assessments[a]—Prophylactic Treatment

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N = 108	EXOSURF N = 107	Placebo (Air) N = 193	EXOSURF N = 192	Placebo (Air) N = 222	EXOSURF N = 224	EXOSURF 1 Dose N = 356	EXOSURF 3 Doses N = 360
	% of Infants		% of Infants		% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)								
Overall	51	57	31	27	36	36	38	35
Severe IVH	26	25	10	8	13	14	9	9
Pulmonary Air Leak (PAL)								
Overall	52	45	16	11	32	25	29	27
Pneumothorax	23	10*	5	6	19	11*	14	12
Pneumopericardium	1	4	2	0	<1	1	1	1
Pneumomediastinum	2	1	2	3	7	1**	3	2
Pulmonary Interstitial Emphysema	43	44	13	7*	26	20	23	22
Death from PAL	4	6	<1	<1	2	1	2	1
Patent Ductus Arteriosus	49	53	66	70	50	55	59	57
Necrotizing Enterocolitis	2	4	11	13	3	4	6	2*
Pulmonary Hemorrhage	2	10**	2	4	1	4	4	6
Congenital Pneumonia	4	4	2	4	2	2	1	1
Nosocomial Pneumonia	10	10	2	4	4	7	14	15
Non-Pulmonary Infections	33	35	34	39	28	29	35	34
Sepsis	30	34	30	34	23	24	30	27
Death From Sepsis	4	4	3	3	1	2	3	2
Meningitis	4	6	3	1	2	3	1	2
Other Infections	7	4	5	3	6	10	10	11
Major Anomalies	3	1	2	4	7	4	4	4

Table 3 Safety Assessments[a]—Prophylactic Treatment (Cont.)

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		1 vs 3 Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N = 108	EXOSURF N = 107	Placebo (Air) N = 193	EXOSURF N = 192	Placebo (Air) N = 222	EXOSURF N = 224	EXOSURF 1 Dose N = 356	EXOSURF 3 Doses N = 360
	% of Infants		% of Infants		% of Infants		% of Infants	
Hypotension	70	77	52	47	59	62	54	50
Hyperbilirubinemia	22	21	63	61	27	31	20	21
Exchange Transfusion	4	3	1	2	2	2	3	1
Thrombocytopenia[b]	21	25	not available		9	8	12	10
Persistent Fetal Circulation	0	1	1	1	0	2*	1	<1
Seizures	11	8	2	2	11	9	6	5
Apnea	34	33	76	73	55	65*	62	68
Drug Therapy								
Antibiotics	96	99	98	96	98	99	>99	99
Diuretics	55	60	39	37	59	63	64	65
Anticonvulsants	14	18	23	24	20	16	9	8
Inotropes	46	40	20	20	26	20	28	27
Sedatives	62	71	65	64	63	57	52	52
Pancuronium	19	11	22	14*	19	13*	15	11
Methylxanthines	38	43	77	77	61	72*	75	82*

[a] All parameters were examined with "as-treated" analyses
[b] Thrombocytopenia requiring platelet transfusion
* p<0.05
** p<0.01

Table 4 Safety Assessments[a]—Rescue Treatment

Number of Doses / Birth Weight Range	Two Doses 700 to 1350 grams		Two Doses 1250 grams and above	
Treatment Group / Number of Infants	Placebo (Air) N = 213	EXOSURF N = 206	Placebo (Air) N = 622	EXOSURF N = 615
	% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)				
Overall	48	52	23	18*
Severe IVH	13	9	5	4
Pulmonary Air Leak (PAL)				
Overall	54	34***	30	18***
Pneumothorax	29	20*	20	10***
Pneumopericardium	4	1	1	2
Pneumomediastinum	8	4	5	2**
Pulmonary Interstitial Emphysema	48	25***	24	13***
Death from PAL	7	3	<1	1
Patent Ductus Arteriosus	66	57	54	45*
Necrotizing Enterocolitis	3	3	1	2
Pulmonary Hemorrhage	3	1	<1	1
Congenital Pneumonia	2	3	2	2
Nosocomial Pneumonia	5	7	2	2
Non-Pulmonary Infections	19	22	13	13
Sepsis	15	17	8	8
Death From Sepsis	<1	<1	1	<1
Meningitis	1	<1	1	<1*
Other Infections	5	8	5	6
Major Anomalies	3	3	4	4
Hypotension	62	57	50	39**
Hyperbilirubinemia	17	19	12	10
Exchange Transfusion	3	4	1	2
Thrombocytopenia[b]	10	11	4	<1**
Persistent Fetal Circulation	1	1	6	2**
Seizures	10	10	6	3*
Apnea	48	65**	37	44*
Drug Therapy				
Antibiotics	100	99	98	98
Diuretics	60	65	45	34***
Anticonvulsants	17	17	10	5**
Inotropes	36	31	27	16***
Sedatives	72	68	76	64***
Pancuronium	34	17**	33	15***
Methylxanthines	62	74**	49	53

[a] All parameters were examined with "as-treated" analyses
[b] Thrombocytopenia requiring platelet transfusion
* p<0.05
** p<0.01
*** p<0.001

Pulmonary Hemorrhage. See WARNINGS. **Abnormal Laboratory Values.** Abnormal laboratory values are common in critically ill, mechanically ventilated, premature infants. A higher incidence of abnormal laboratory values in the Exosurf Neonatal group was not reported. **Events During Dosing:** Data on events during dosing are available from more than 8800 infants in the open, uncontrolled clinical study (Table 5).

Table 5 Events During Dosing in the Open, Uncontrolled Study[a]

Treatment Type / Number of Infants	Prophylactic Treatment N = 1127	Rescue Treatment N = 7711
	% of Infants	% of Infants
Reflux of Exosurf	20	31
Drop in O_2 saturation (≥ 20%)	6	22
Rise in O_2 saturation (≥ 10%)	5	6
Drop in transcutaneous pO_2 (≥ 20 mm Hg)	1	8
Rise in transcutaneous pO_2 (≥ 20 mm Hg)	2	5
Drop in transcutaneous pCO_2 (≥ 20 mm Hg)	<1	1
Rise in transcutaneous pCO_2 (≥ 20 mm Hg)	1	3
Bradycardia (<60 beats/min)	1	3
Tachycardia (>200 beats/min)	<1	<1
Gagging	1	5
Mucous Plugs	<1	<1

[a] Infants may have experienced more than one event.
Investigators were prohibited from adjusting FiO_2 and/or ventilator settings during dosing unless significant clinical deterioration occurred.

Reflux. Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes. ***Mucous Plugs.*** See WARNINGS.

DOSAGE AND ADMINISTRATION See full product information.

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for NEB therapy can be accomplished with considerable, but not total, success. This approach results in significant reductions in the cost of health care provision.

The Prehospital Treatment of Pediatric Trauma—RF Lavery, BJ Tortella, CC Griffin. Pediatr Emerg Care 1992;8:9.

Prehospital pediatric trauma care is an important part of the EMS system. Review of 458 pediatric ALS trauma responses over 2 years treated in an urban, tiered ALS system revealed a male predominance. Violence (gunshot, stab, or assault) accounted for 46% of injuries, followed by vehicular accidents (occupant or pedestrian) with 35%. Important ALS resuscitation interventions were commonly performed en route, with a high degree of success (I.V.s = 93%, intubation = 79%), and did not greatly prolong field times (9 min BLS vs 11.7 min ALS). ALS procedure success rates and field times reported here are lower than previously described. Benchmark standards for the prehospital care of pediatric trauma are proposed.

Pediatric EMS Transport: Are We Treating Children in a System Designed for Adults Only?—A Sacchetti, C Carraccio, M Feder. Pediatr Emerg Care 1992;8:4.

Unlike adults, small children and infants do not require stretchers or ambulances for transport from a prehospital scene to the emergency department (ED). This study was designed to determine the importance of this difference in patient transport needs. A Macintosh Classic computer was programmed to compare the time to intubation (TTI) of a child with impending respiratory arrest treated in a standard paramedic/ambulance transport system versus that of a child treated in a system in which a patrolling police car transports the child directly to an ED. The dependent variable TTI was determined, with travel times from the scene to the ED and paramedical intubation success rates as the independent variables. Utilizing this model, police transports demonstrate shorter TTIs for brief scene-to-ED travel times or limited paramedic success rates, while paramedic intubations produced shorter TTIs for long scene-to-ED transports. These results suggest that nonambulance transport of pediatric patients be considered in the development of urban or suburban pediatric Emergency Medical Services.

Pilot Study for the Development of a Monitoring Device for Ventilated Children—DF Wensley, P Noonan, MD Seear, H Werner, GE Pirie. Pediatr Pulmonol 1991;11:272.

SUMMARY: Airway pressure and air flow were measured at the endotracheal tube in 13 children on a variety of ventilators. These signals were stored for analysis on a computer. Further data sets were obtained after 24 hours or following major interventions. Air flow rate was integrated to give volume. Pulmonary resistance and elastance were obtained by multiple linear regression. Pressure-volume, pressure-flow, and flow-volume loops were plotted. "Closed" pressure-volume and pressure-flow loops (by subtraction of the resistive or elastic pressure components, respectively) were also displayed, giving compliance and resistance loops. The loops from the initial data set were taken as the baseline, and loops from later data sets were superimposed to provide visual comparisons. Change in clinical status was reflected by the change in slope of compliance and resistance loops. A 30% change in compliance or resistance was easily observed There was minimal interference with patient care. This pilot study demonstrates that changes in respiratory mechanics can be displayed safely and easily in ventilated patients using resistance and compliance loops. Further work is necessary to confirm the usefulness of real time of these displays.

Comparison of Six ECMO Selection Criteria and Analysis of Factors Influencing Their Accuracy—NR Payne, P Kriesmer, M Mammel, CL Meyer. Pediatr Pulmonol 1991; 11:223.

SUMMARY: This study compared six extracorporeal membrane oxygenation (ECMO) selection criteria in 42 neonates and analyzed factors influencing the accuracy of outcome predictions. The sensitivity of the criteria in identifying fatal cases varied from 0.44 to 0.94 and the specificity of predictions of survival ranged from 0.42 to 0.69. The criterion having the highest sensitivity had the lowest specificity and conversely the criterion with the lowest sensitivity had the highest specificity. Overall accuracy of the criteria, as measured by the total number of correct outcome predictions, differed little among the criteria (23/42 to 27/42 correct predictions). Three factors influenced predictive accuracy: (1) a primary diagnosis of congenital diaphragmatic hernia (CDH) was associated with a greater mortality ($p < 0.001$) and a significantly higher positive predictive value (PPV) for all criteria ($p = 0.0009$-0.012) than that seen in patients with other primary diagnoses; (2) calculating the alveolar-arterial oxygen gradient using an assumed, rather than measured barometric pressure, or estimating oxygenation index using a calculated, rather than a measured, mean airway pressure, increased false-positive mortality predictions in non-CDH patients; and (3) requiring a peak inspiratory pres-

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sure (PIP) of at least 50 cm H_2O in the definition of maximal medical management, rather than a PIP of 20-49 cm H_2O, significantly increased the PPV for three of four criteria examined ($p = 0.02$-0.04). Awareness of these factors may facilitate the identification of neonates who need ECMO to survive.

Nocturnal Positive-Pressure Ventilation via Nasal Mask in Patients with Severe Chronic Obstructive Pulmonary Diseases—DA Strumpf, RP Millman, CS Carlisle, LM Grattan, SM Ryan, AD Erickson, NS Hill. Am Rev Respir Dis 1991;144: 1234.

SUMMARY: Intermittent positive pressure ventilation administered nocturnally via a nasal mask has been associated with improvements in pulmonary function and symptoms in patients with restrictive ventilatory disorders. We hypothesized that nocturnal nasal ventilation (NNV) would bring about similar improvements in patients with severe chronic obstructive pulmonary disease (COPD). The study used a randomized, crossover design, with subjects undergoing NNV or "standard care" for sequential 3-mo periods. Of 23 patients with obstructive lung disease and a $FEV_1 < 1$ L who were initially enrolled, 4 were excluded because of obstructive sleep apnea prior to randomization. Among the remaining 19 patients, 7 withdrew because of intolerance of the nose mask, 5 were withdrawn because of intercurrent illnesses, and 7 completed both arms of the protocol. These latter 7 patients used the ventilator for an average of 6.7 h/night, and 3 of the 7 had partial relief of dyspnea during ventilator use. However, in comparison with studies performed upon initiation or after the standard care arm of the study, studies performed after 3 months of NNV revealed no improvement in pulmonary function, respiratory muscle strength, gas exchange, exercise endurance, sleep efficiency, quality or oxygenation, or dyspnea ratings. The only improvements observed were in neuropsychological function, possibly related to a placebo effect or another unknown mechanism Despite the small sample size, our study indicates that NNV is not well tolerated by and brings about minimal improvement in stable outpatients with severe COPD.

Airway Insufflation: Increasing Flow Rates Progressively Reduce Dead Space in Respiratory Failure—AN Hurewitz, EH Bergofsky, E Vomero. Am Rev Respir Dis 1991; 144:1229.

SUMMARY: We have previously shown that airway insufflation (AI) reduces dead space (V_D) and minute ventilation ($\dot{V}_L$) in patients with respiratory failure, and when used chronically leads to lowered and more stable arterial P_{CO_2}. The present study was designed to measure the effect of increasing AI flow rate on V_D and other aspects of gas exchange in respiratory failure in order to examine the hypothesis that AI exerts its main physiologic effect by progressive reductions of V_D. Five patients with varying degrees of respiratory failure caused by either restrictive or obstructive lung disease were studied by means of the specialized techniques we developed to analyze gas exchange during AI. At 1 L/min (as in transtracheal oxygenation), at 5 L/min, and 8 L/min, AI produced progressive reductions in V_D, tidal volume, and $\dot{V}_L$. Contrary to our previous study, some of these patients accompanied the decrease in V_D with not only decreases in $\dot{V}_L$ but with slight rises in alveolar ventilation (V_A) and decrements in arterial P_{CO_2}. The greatest percentile decreases in V_D and $\dot{V}_L$ occurred in those with the smallest initial control values for each of these parameters. In summary, AI exerts its main effects on gas exchange through the reductions in V_D that it produces, and the accompanying decreases in $\dot{V}_L$ and/or slight increases in V_A seem to stem from the latter.

Theophylline and Salbutamol Improve Pulmonary Function in Patients with Irreversible Chronic Obstructive Pulmonary Disease—P Thomas, JA Pugsley, JH Stewart. Chest 1992;101:160.

To investigate the efficacy of bronchodilators in patients with irreversible chronic obstructive pulmonary disease (COPD), we conducted a double-blind, randomized, four-phase, crossover comparison between placebo, oral theophylline, inhaled salbutamol, and a combination of both drugs in 12 patients with stable COPD (mean age, 63 years) whose increase in forced expiratory volume in 1 s (FEV_1) was ≤ 15% following 200 μg of inhaled salbutamol. Patients received 2 weeks of therapy with each of the test regimens. Both theophylline and salbutamol resulted in statistically significant improvement in FEV_1, forced vital capacity (FVC), slow vital capacity (SVC), residual volume (RV), airway resistance (R_{aw}), and maximum expiratory flow rate at 50% of vital capacity ($\dot{V}_{50}$). In most instances, there were no significant differences between theophylline and salbutamol. Combination therapy produced significantly greater improvement in FEV_1, FVC, $\dot{V}_{50}$, R_{aw}, and RV than either agent alone. The two drugs interacted in an additive fashion. Neither of the drugs, used singly, significantly reduced the severity or incidence of symptoms. The reduction in dyspnea and wheeze during combination therapy ap-

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INDICATIONS AND USAGE

SURVANTA is indicated for prevention and treatment ("rescue") of Respiratory Distress Syndrome (RDS) (hyaline membrane disease) in premature infants. SURVANTA significantly reduces the incidence of RDS, mortality due to RDS and air leak complications

Prevention

In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth

Rescue

To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age

CONTRAINDICATIONS

None known.

WARNINGS

SURVANTA is intended for intratracheal use only

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants The causative organisms were similar in treated and control infants There was no significant difference between groups in the rate of post-treatment infections other than sepsis

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed Mutagenicity studies were negative Carcinogenicity studies have not been performed with SURVANTA

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of doses Oxygen desaturation occurred with 9 8% of doses.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials The rates in all controlled studies are in Table 3

TABLE 3

	All Controlled Studies		
Concurrent Event	SURVANTA (%)	Control (%)	*P*-Value[a]
Patent ductus arteriosus	46 9	47 1	0 814
Intracranial hemorrhage	48 1	45 2	0 241
Severe intracranial hemorrhage	24 1	23 3	0 693
Pulmonary air leaks	10 9	24 7	<0 001
Pulmonary interstitial emphysema	20 2	38 4	<0 001
Necrotizing enterocolitis	6 1	5 3	0 427
Apnea	65 4	59 6	0 283
Severe apnea	46 1	42 5	0 114
Post-treatment sepsis	20 7	16 1	0 019
Post-treatment infection	10 2	9 1	0 345
Pulmonary hemorrhage	7 2	5 3	0 166

[a]*P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63 3% *v* 30 8%, $P = 0.001$, and 48 8% *v* 34 2%, $P = 0.047$, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests white blood cell count and serum sodium, potassium, bilirubin, creatinine

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C No IgG or IgM antibodies were detected

Several other complications are known to occur in premature infants The following conditions were reported in the controlled clinical studies The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer

Renal: renal failure, hematuria

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia

Musculoskeletal: inguinal hernia

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results

OVERDOSAGE

Overdosage with SURVANTA has not been reported Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present

HOW SUPPLIED

SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0 9% sodium chloride solution The color is off-white to light brown

Store unopened vials at refrigeration temperature (2-8°C) Protect from light Store vials in carton until ready for use. Vials are for single use only Upon opening, discard unused drug

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proached statistical significance ($p = 0.06$) and patient preference was significantly in favor of the combination regimen. None of the active treatments produced significantly more side effects than placebo. We conclude that theophylline and inhaled salbutamol produce significant, and approximately equal, improvement in pulmonary function in patients traditionally classified as suffering from "irreversible" COPD. The combination of theophylline and inhaled salbutamol generally results in additional improvement over that obtained with either drug used alone and this improvement is reflected by reduced symptomatology and treatment preference.

Utility of the Peak Expiratory Flow Rate in the Differentiation of Acute Dyspnea: Cardiac vs Pulmonary Origin—RM McNamara, DJ Cionni. Chest 1992;101:129.

This study examined the utility of a peak expiratory flow rate (PEFR) measurement in the differentiation of acute moderate-to-severe dyspnea secondary to congestive heart failure or chronic lung disease. A PEFR was determined in 41 episodes of acute respiratory distress in 40 patients prior to emergency department therapy. The mean PEFR ± SD for the congestive heart failure group ($n = 18$) was 224 ± 82 L/min, which was significantly higher ($p < 0.001$) than that of the chronic lung disease group ($n = 23$), which had a mean PEFR of 108 ± 49 L/min. No single cutoff value allowed 100% accurate classification, but the results suggest that the PEFR may be a useful adjunctive tool in the differentiation of acute dyspnea of cardiac vs pulmonary origin.

Health Care Workers Infected with the Human Immunodeficiency Virus: The Next Steps—B Lo, R Steinbrook. JAMA 1992;267: 1100.

The tragedy of five patients who contracted human immunodeficiency virus (HIV) infection from a seropositive dentist has alarmed the public. The Centers for Disease Control (CDC) recently revised its recommendations for preventing the transmission of HIV infection to patients during invasive procedures. The CDC abandoned a previous plan to list exposure-prone invasive procedures that HIV-infected health care workers should not perform. The CDC said "expert review panels" should decide on a case-by-case basis whether seropositive health care workers may perform invasive procedures. As of February 1992, the revised recommendations were under review by the U.S. Department of Health and Human Services. Many issues remain to be clarified, such as how these panels will operate and whether decisions will be consistent in similar cases. Disregarding the CDC guidelines or infection-control precautions may further erode public trust and lead to draconian restrictions on HIV-infected health care workers. Physicians and dentists should respond more effectively to public fears about HIV transmission. The challenge is to protect patients while respecting the privacy and livelihood of health care workers.

Complications of Endotracheal Intubation and Mechanical Ventilation in Infants and Children—R Rivera, J Tibballs. Crit Care Med 1992;20:193.

OBJECTIVE: To assess the frequency of complications of endotracheal intubation and mechanical ventilation. DESIGN: Prospective cohort study. SETTING: Pediatric tertiary multidisciplinary ICU. PATIENTS: Eight hundred sixty-nine serial patients, of whom 500 were endotracheally intubated and 431 were mechanically ventilated. INTERVENTIONS: Daily clinical observation. MAIN OUTCOME MEASURES: Age, diagnosis, endotracheal tube size, type of ventilator and humidification, duration of intubation and mechanical ventilation, ventilatory settings, and complications (accidental extubation, tissue damage, endobronchial intubation, postintubation stridor, endotracheal tube blockage, pulmonary airleak, bronchopulmonary dysplasia, pulmonary

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atelectasis) were recorded. RESULTS: The median patient age was 10 months (25th and 75th quartiles: 1 month and 15 yrs). The median duration of endotracheal intubation and mechanical ventilation was 4 days and 2 days, respectively. A total of 186 complications were recorded in 119 (24%) patients. No deaths were due to intubation or ventilation. The overall frequency of accidental extubation was 3.2% of patients at a rate of 1.26 extubations/100 intubation days. In nonventilated, unsedated patients with croup or epiglottitis, the frequency of accidental extubation was 23% at a rate of 11.83 extubations/100 intubation days. Lung atelectasis occurred in 7.8%, airleak in 6.8%, and infection in 2.3% of patients. Other complications included tissue damage (3.4%), endobronchial intubation (2.4%), postintubation stridor (2.4%), bronchopulmonary dysplasia (2.3%), and endobronchial tube blockage (0.8%). CONCLUSIONS: Complications were related to the duration of intubation and mechanical ventilation.

Benefits of a Lower Hematocrit during Extracorporeal Membrane Oxygenation?—MP Griffin, PK Minifee, CW Daeschner III, JB Zwischenberger. AJDC 1992;146:373.

OBJECTIVE: To determine the possible benefits of maintaining a lower hematocrit than that normally used (0.35 vs 0.45) in neonates treated with extracorporeal membrane oxygenation. DESIGN: Randomized cohort. SETTING: Neonatal and pediatric intensive care units at a university hospital. PARTICIPANTS: Twenty neonates who met criteria for receiving extracorporeal membrane oxygenation from May 1988 to March 1990. INTERVENTIONS: Hematocrits were maintained at 0.35 for neonates in Group 1 and 0.45 for neonates in Group 2. MEASUREMENTS/MAIN RESULTS: Hematocrits were measured every 4 hours. Visible clots in the major circuit components were recorded. Infants in Group 1 received (mean ± SD) 2.5 ± 0.6 mL of packed red blood cells per hour of extracorporeal membrane oxygenation while infants in Group 2 received 3.8 ± 0.9 mL of packed red blood cells per hour of extracorporeal membrane oxygenation. In Group 1, clots were noted in six of 10 oxygenators and five of 10 bladder reservoirs. In Group 2, clots were found in all 10 oxygenators and bladder reservoirs. CONCLUSIONS: Neonates' hematocrits can be maintained safely at 0.35 during extracorporeal membrane oxygenation with significantly less exposure to packed red blood cells and less clotting in the circuit.

Clinical Value of Polysomnography—NJ Douglas, S Thomas, MA Jan. Lancet 1992;339:347.

Polysomnography is used increasingly to investigate patients with possible sleep apnoea/hypopnoea syndrome (SAHS), but it has not been assessed critically. We thus examined prospectively the value of electrophysiological and respiratory monitoring in 200 consecutive adults (163 men, 37 women; mean [SD] age 50 [13] years) having polysomnography. At polysomnography, 91 patients had SAHS (> 15 apnoeas + hypopnoeas [A + H] per h asleep) and 11 had periodic limb-movement disorder. Recording sleep electrophysiologically was of no diagnostic value and SAHS could be as accurately defined by A + H per time in bed as by A + H per time asleep. 66% of patients with SAHS could be diagnosed with oximetry alone, but many of the undiagnosed patients had moderately severe SAHS and benefited from treatment. Neurophysiological sleep recording is unnecessary and oximetry alone is of limited value in the overnight investigation of patients suspected of having SAHS.

An Unsuccessful Cotinine-Assisted Intervention Strategy To Reduce Environmental Tobacco Smoke Exposure during Infancy—BA Chilmonczyk, GE Palomaki, GJ Knight, J Williams, JE Haddow. AJDC 1992;146:357.

OBJECTIVE: To test a low-intensity physician's office-based intervention strategy using infant urine cotinine measurements, aimed at reducing infant exposure to environmental tobacco smoke. DESIGN: A randomized intervention trial. SETTING: Offices of 28 physicians (pediatricians and family practitioners) and two hospital-based clinics in the Greater Portland, Maine, area. PARTICIPANTS: Infants brought to the physician by a parent (usually the mother) for an initial well-child visit. A total of 518 of these infants and their mothers consented to provide information about household smoking habits and to

1992 PUBLICATION AWARDS

See Page 384 for Details

provide a sample of the infant's urine for biochemical analysis. SELECTION PROCEDURE: From among the consecutive sample of 518 enrolled mother/infant pairs, 103 mothers reported that they smoked 10 or more cigarettes per day, and these were randomized on an individual basis for intervention (52 pairs) or control (51 pairs). Randomization took place at the study center when the infant's urine sample and information about household smoking habits were received. INTERVENTION: The physician telephoned the mother to report the urine cotinine result and to explain its meaning. The physician then signed and sent an individualized form letter to the mother, providing specific recommendation for changing household smoking habits. RESULTS: Follow-up urine cotinine measurements were obtained in 27 (52%) of 52 infants from the intervention group and in 29 (57%) of the 51 controls 2 months later. The mean log ratio of the follow-up to initial urine cotinine measurements was 6% lower in the intervention group than in the control group. This difference was not statistically significant. CONCLUSION: The low-intensity intervention strategy did not significantly influence infant exposure to environmental tobacco smoke in the household.

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The Hospitalized COPD Patient: 10 Commandments for the Clinician

By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the ten commandments for the clinician to follow when the COPD patient presents to the hospital. Emphasis is on the "what-not-to-do."
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Pressure Support Update

By Neil MacIntyre, MD. Briefly reviews pressure support ventilation (PSV) and how it is used. Explains waveforms for airway pressure, flow, and volume. Also illustrates the measurement of inspiratory muscle loads by pressure-time index and muscle V_{O_2}, and demonstrates how muscle efficiency changes as the character of work changes. Includes examples of clinical applications of low-level and high-level PSV for elimination of the imposed work of breathing and as a weaning tool.
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Managing the Work of Breathing During Mechanical Ventilation

By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation. This presentation focuses on diminishing the breathing work load through quantifying the effort during mechanical ventilation via direct measures such as oxygen consumption, electromyography, press-time index, and external work of breathing. Also points out indirect measures such as breathing patterns and dyspnea.
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Tissue Oxygen Delivery

By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. Also discusses the factors that govern oxygen transport to the tissues and the variables that reflect the adequacy of tissue oxygen transport Explores microvascular oxygen transport as depicted by the Krogh model versus a model that incorporates tissue shunting and a countercurrent mechanism to explain tissue hypoxia — unpredictable from P_{aO_2}.
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Theory and Application of Neonatal Ventilation

By Robert Chatburn, RRT. Knob turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally

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has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation.
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Can Surfactant Prevent Respiratory Distress Syndrome?

By Alan Jobe, MD, PhD. Research indicates that instillation of surfactant into the lungs of neonates at risk for the development of severe RDS can result in improved lung mechanics. If safe and effective, why is this promising therapeutic regimen not available in most medical centers? Dr. Jobe is one of the nation's leading authorities on the use of replacement surfactant in the treatment/prevention of RDS
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Fetal Lung Development

By Charles Rosenfeld, MD. This presentation examines the four anatomical phases of fetal lung development which is followed by a discussion of the biochemical development of the fetal lung through gestation. Includes a description of the substances in surfactant and the importance of their timely development. Surfactant function is described along with the methods used to assess fetus survivability by using tracheal aspirant to identify the necessary ratios of the phospholipids making up surfactant.
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Clinical Use of the Swan-Ganz Catheter

By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter (SGC) placement and the situations in which SGC placement are useful are described in this video. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement.
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Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak

By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function (e.g., pneumothorax, as well as one-, two-, and three-bottle chest drainage systems). Discussion covers the potential problems associated with chest tubes stripping, difficulties of bronchopleural air leaks with mechanical ventilation, the therapeutic goals of chest tube placement, and techniques for maintaining gas exchange with air leaks.
Item VT21 — $40 Ea, VHS (Member $35)

Clinical Prediction and Prevention of ARDS

By Leonard D. Hudson, MD. The mortality of patients diagnosed with adult respiratory distress syndrome has remained unchanged since 1976. This tape investigates a system of dealing with potential ARDS patients that may decrease mortality. The discussion centers on the identification of patients at risk of developing ARDS, their risk factors, and risk criteria. An approach to detect and prevent ARDS is also presented.
Item VT20 — $40 Ea, VHS (Member $35)

Modes of Conventional Ventilation

By Robert M. Kacmarek, PhD, RRT. A review of mechanical ventilation is presented, including the rationale for movement from one generation of ventilators to another. Ventilator modes discussed include control, assist/control, intermittent mandatory ventilation, synchronized intermittent mandatory ventilation, mandatory minute ventilation, and pressure support ventilation. Description includes typical pressure waveforms of each mode.
Item VT19 — $40 Ea, VHS (Member $35)

Pressure Support Ventilation

By Neil MacIntyre, MD. This presentation defines and describes the physiological considerations attributed to PSV and the clinical situations when PSV may be useful. Includes comparisons when low pressure levels and high pressure levels of PSV are offered. Discussion includes using PSV to help overcome resistance for intubated patients and as an augmented ventilatory mode of weaning. Included are results of patient weaning trials with and without PSV.
Item VT17 — $40 Ea, VHS (Member $35)

Practical Management of ARDS

By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape and its clinical features described, including incidence of risk factors and clinical predictors. Also extensively discussed is the use of PEEP to treat ARDS, including goals, complications, best or optimal PEEP levels, PEEP trials, and PEEP withdrawal as well as general treatment, prognosis, and sequelae of ARDS.
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By Jon Weissler, MD. A historical perspective of acquired immune deficiency syndrome is presented, including epidemiological considerations, demographics, and social ramifications. Instruction is also provided on the pathogenesis of AIDS with emphasis on diagnosis and treatment of pulmonary manifestations.
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Sleep Apnea

By Alan K. Pierce, MD. Explains how sleep stages, as recorded by electroencephalography, are related to respiratory patterns and blood gas values in both normal and abnormal subjects. Includes a discussion of the criteria for defining the sleep apnea syndrome and the distinguishing features of central, obstructive, and mixed causes of apnea. Also addressed is the efficacy of medical treatment to correct specific types of sleep apnea.
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The Effect of Respiratory Care Department Management of a Blood Gas Analyzer on the Appropriateness of Arterial Blood Gas Utilization

Kathryn E Beasley RRT, James M Darin RRT, and Charles G Durbin Jr MD

We investigated the effects of establishing a blood gas analysis service controlled by respiratory care practitioners (RCPs) on the appropriateness of arterial blood gas (ABG) sampling. An ABG analyzer was placed outside the surgical intensive care unit (SICU) and only RCPs were permitted to process samples on it. In 1-month and 1-year follow-up audits of appropriateness of ABG analysis, the nursing staff improved from 42% appropriate to 73% appropriate in both follow-up periods. RCPs maintained a high degree of appropriateness in all periods (90%, 87%, and 91%), although the percentage of the total ABGs performed by RCPs increased. Additional benefits included a better mutual understanding of each caregiver's role and work load, more collaboration among caregiver groups, and caregivers' perception of improved patient care. (Respir Care 1992;37:343-347.)

Introduction

Analysis of arterial blood to assess the respiratory status of critically ill patients is widely practiced and well accepted. Repeated measurements providing information about trends in arterial blood gas (ABG) values—oxygen and carbon dioxide gas exchange and acid-base balance—are standard in the intensive care unit. Frequent measurements are made to monitor the progress of disease, assess effects of therapy, adjust medications, identify patients at risk of pulmonary compromise, and help caregivers make decisions regarding respiratory and other intensive care support.

Although many benefits may result from routine and frequent measurements of ABG values, these measurements are often used for surveillance monitoring. The advent of noninvasive techniques for trending respiratory data (such as capnography and pulse oximetry) has diminished the need for frequent, repetitive ABG determinations. In a previous study, we showed that education of caregivers in deciding when to draw arterial samples reduced the frequency of unnecessary ABG analysis. After developing an algorithm based on specific criteria, and educating physicians, nurses, and respiratory therapists about its use, we were able to reduce the unnecessary blood gas analyses from 42.7% to 30.8% in a surgical intensive care unit (SICU).[1] This effect persisted over at least the following year. When we identified who was obtaining the "inappropriate" ABG analyses, we found that the inappropriate rate for RCPs was 15% while the so-called others group (primarily nurses and physicians) was 37%.

We were presented with a new investigative opportunity while continuing an intensive orientation and education process when, in the Spring of 1989, the SICU moved into a new building. Prior to the move, ABG analysis had been provided by a 'stat' clinical laboratory immediately adjacent to the SICU. During this time, results were available

Ms Beasley was a Respiratory Therapist B when this work was done, Mr Darin is Coordinator, Adult Surgical Critical Care, and Dr Durbin is Medical Director—Department of Respiratory Care, University of Virginia Health Sciences Center, Charlottesville, Virginia. Ms Beasley is now a respiratory therapist at Good Samaritan Hospital, Dayton, Ohio.

A version of this paper was presented by Ms Beasley during the RESPIRATORY CARE OPEN FORUM at the 1991 AARC Annual Meeting in Atlanta, Georgia.

Reprints: Charles G Durbin Jr MD, Department of Respiratory Care, Box 402, University of Virginia Health Sciences Center, Charlottesville VA 22908.

from the dedicated laboratory within 5 minutes. In moving to the new hospital, the laboratories were separated from the SICU by 4 floors. This required an off-unit messenger to transport the sample from the SICU to the laboratory; the average turnaround time for ABG analysis was 37 minutes.

We hypothesized that when ABG analysis was very much delayed, routine surveillance use of analysis would decrease. In cases when patients required more rapid analysis, the nurse or other personnel from the unit would walk (run) the sample to the laboratory, thus obtaining results in approximately 10 minutes.

As part of our Quality Improvement (QI) program, we continually monitor the appropriateness of ABG analysis according to the previously described guidelines. Contrary to our hypothesis, we found no improvement in the appropriateness of ABG utilization with the inconvenience of the distant laboratory.

Due to clinician concern for the lack of availability of rapid laboratory analysis in critical patients needing such care, a Corning 282 ABL blood gas analyzer (Corning Automated Blood Gas Laboratory 282, Ciba-Corning, Medfield MA) was placed immediately outside the SICU. RCPs trained in its use were the only personnel permitted to process ABG samples. Analyzer quality control and maintenance was provided daily by clinical laboratory personnel. Because the lone, unit-based RCP was required to leave the SICU to analyze the ABG, we formulated the hypothesis: A forced interaction between SICU RCPs and nurses over the 'need' for a specific ABG analysis will decrease the number of ABG samples drawn and increase the appropriateness of sampling.

In the event that the RCP and nurse disagreed over the need for a blood gas analysis, the nurse could still send the sample to the main laboratory with the usual prolonged turnaround time.

Methods

Appropriateness of ABG analysis was studied during 3 periods: prior to introduction of the ABG analyzer, 1 month following its introduction, and 1 year later. Approximately 150 ABGs were sampled from randomly selected patients representing each working shift for each study period. Each patient's critical care flow sheet was examined at the time an ABG sample was drawn to determine the clinical events surrounding the decision to draw the sample. The previously described ABG algorithm was utilized to determine appropriateness.[1] The criteria in this algorithm are based on procedural, clinical, and therapeutic indicators. Procedural indicators include recent admission to the SICU, high levels of ventilatory support, and anticipation of tracheal extubation. Clinical indicators include signs and symptoms of hypoxia, hypercarbia, and increased work of breathing. Therapeutic indicators include prior ABG abnormalities, changes in respiratory support, and changes in bronchodilatory or vasoactive medications. A Yes answer to any of the following questions is considered justification of the need for an ABG analysis.

Procedural indicators:

1. Is the patient a new admission to the SICU?
2. Is the F_{IO_2} more than 0.60 and has it been 3 or more hours since the previous ABG analysis?
3. Is the patient on PEEP/CPAP of more than 10 cm H_2O and has it been 3 or more hours since the previous ABG analysis?
4. Is there an order to extubate the patient within the next 30 minutes?

Clinical indicators:

1. Does the patient exhibit any of the following?
   a. absence of breath sounds?
   b. asynchronous breathing?
   c. cyanosis?
   d. diaphoresis?
   e. pallor?
   f. unexpected dysrhythmia?
   g. unexpected change in mental status?
2. Has there been an unexpected change of 20% in respiratory rate, or is the spontaneous + ventilator rate > 35 or < 5 breaths/minute?
3. Has there been an unexpected change of 30% in heart rate?
4. Has there been an unexpected change of 30% in systolic blood pressure?
5. Has there been an unexpected change of 20% in cardiac output?
6. Has there been an unexpected and sustained increase of > 5 torr in ICP (intracranial pressure) or an absolute ICP of > 20 torr?
7. Does the patient's previous ABG report have any of the following?
   a. $P_{aO_2} < 60$ or > 125 torr?
   b. Measured $S_{aO_2} < 85\%$?
   c. $P_{aCO_2} > 65$ or < 20 torr?
   d. pH > 7.55 or < 7.30?

Therapeutic indicators:

1. Has there been an extubation or a change in a respiratory support variable (oxygen concentration, tidal volume, rate, PEEP/CPAP, mode of delivery)?
2. Has there been a change in bronchodilator or vasoactive medication?

For an ABG analysis to be deemed appropriate, it had to meet only one of these criteria. It is important to remember that it is not necessarily wrong or inappropriate to draw an arterial sample if these criteria are not met; however, these were the guidelines used in this study. Inadequate flow-sheet documentation could result in an appropriate sample being labeled inappropriate.

After each of the ABG analyses had been determined to be either appropriate or inappropriate, the category of caregiver who drew the sample was recorded (RCP, nurse, or unknown). The percentage of appropriate and inappropriate analyses by personnel in each category and the number of ABG analyses per patient-day were calculated. We used chi-square analysis in each of the caregiver categories to compare performance during each of the time periods.

Results

Appropriateness of ABG Analysis by Caregiver Category

Table 1 shows the results of the evaluation of the appropriateness of randomly selected ABG analyses from Period I (the control period, prior to installation of the SICU analyzer), Period II (the month following its installation), and Period III (1 year after installation). The percentage of appropriate analyses for nurses and RCPs is shown in Figure 1. Chi-square analysis (p values indicated) indicates significant improvement in frequency of appropriateness of ABGs initiated by nurses in each of the follow-up periods compared to the control period but no change in practice by RCPs.

Table 1. Number and Percentage of Arterial Blood Gas Analyses Obtained by Respiratory Care Practitioners, Nurses, and Uncategorized Personnel that Met at least One Criterion for Appropriateness

Caregiver	Period I (Prior to Installation)	Period II (1 Month after Installation)	Period III (1 Year after Installation)
RCP	17/19 (90%)	67/72 (93%)	32/35 (91%)
RN	23/55 (42%)	36/49 (75%)	61/84 (75%)
Category unknown	47/74 (64%)	26/29 (90%)	20/31 (65%)
Total	87/148 (59%)	120/150 (86%)	113/150 (75%)

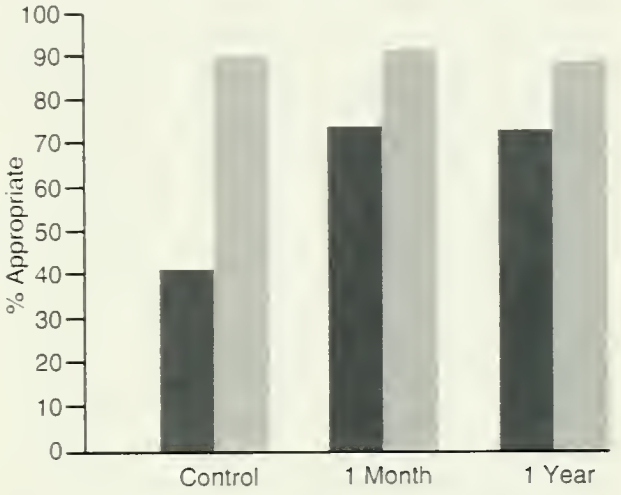

Fig. 1. Percentage of arterial blood gas analyses meeting "appropriateness" criteria for nurses ■ and respiratory care practitioners □ in the control and two follow-up periods. Values in follow-up periods were compared to control values by chi-square analysis to establish p values (1 month, $p = 0.035$; 1 year, $p = 0.066$). No significant change was seen in the appropriateness of the RCP group in any period.

Analyses per Patient-Day

During the control period 3.8 ABGs were obtained per patient-day, during the first review period 3.1 were obtained per patient-day, and during the period 1 year later 3.7 ABGs per patient-day were obtained. These differences were not significant.

Discussion

We have shown in this intensive care unit environment that introduction of RCP-managed ABG analysis can produce an immediate and sustained improvement in the appropriateness of ABG analysis initiated by other care practitioners. Total concordance of appropriateness between nursing and

RCP-initiated ABGs was not possible due to the availability of an alternative route to the main laboratories for analysis.

Frequency of ABG analysis was not reduced in this study. Thoughtful decision making and discussion of patient clinical status may have led caregivers to obtain some samples that might otherwise not have been obtained. The fact that more of the ABG analyses performed met a criterion considered appropriate is of primary importance. We believe that the forced educational and collaborative interaction between RCPs and nurses was the main reason for improvement in decision making regarding ABG analysis.

Although we saw no reduction in frequency of sampling and thus in the amount of blood removed from patients, reduction of blood loss is another motivation for reducing the number of ABG analyses performed each day. At times, blood loss from sampling can be important and is often underestimated.[2] This can be a contributor to the need for transfusion with attendent risks. We did not decrease the sampling rate (ie, ABGs/patient-day) with this intervention. Patient severity of illness and intensity of treatment are important determinants of frequency of ABG sampling and may have been greater in the follow-up periods.

Failure to show a reduction in the ABG analysis rate may also be a function of the way this number was obtained during the study. Patients representing all working shifts were selected at random during the test periods, and all ABGs obtained in the selected patients evaluated. When a total of 150 analyses were identified, the number of patient-days that were needed to obtain these was used as the denonimator. In general, this consisted of only 8 to 10 patients in each test period. The number of ABG analyses per patient varied widely. The small number of patients did not represent all of the diseases in the unit. A better evaluation of the effect of this change in practice on sampling rate would be to increase the size of the sample or independently monitor the number of blood gases per patient-day. We plan to do this as well as estimate the severity of patient illness by including in a future study the total number of ventilator hours per patient-day during the test periods.

One significant area not addressed by this study or the algorithm is, When is an ABG analysis indicated but not obtained? As the algorithm is weighted toward clinical indications, it may stimulate more frequent (appropriate) analyses. This thoughtful, rather than 'routine' use of laboratory studies may lead to better patient care, more rapid ventilator weaning, and shorter hospital stay. This is certainly the goal of improving care, but we have no direct evidence of this happening during this study. Others have reported improved efficacy and quality of care by modifying caregivers' use of laboratory studies by various methods.[3,4]

A by-product of this collaborative effort was that relationships between RCPs and nurses improved during this time period. The team approach to deciding when ABG analysis was appropriate helped build trust related to other aspects of responsibility as well. When the RCP was busy with acute patient care, the nurse would not draw an 'unnecessary' sample, but might wait until both she and the RCP could evaluate the patient together. In our SICU, the lone RCP is responsible for 4 to 7 ventilated patients, while the nurse has, at most, 2 patients for whom to care. The usual nursing concern that the RCP is "not around" (ie, he/she is not always at his/her patient's bedside) soon vanished as appreciation for the RCP's work load rose.

We believe that education of new RCPs, nurses, and physician staff is important to maintain the improvement in ABG sampling we obtained. This belief is supported by a study by Bareford and Hayling, who found that with education, provision of guidelines, and positive feedback, as much as a 38% reduction was seen in the number of inappropriate requests for laboratory services.[5]

As part of our QI program, we present the ongoing results of this assessment on a periodic basis to respiratory, physician, and nursing staffs. Improvements are announced publicly on bulletin boards within the unit. Competition to improve appropriateness of laboratory utilization between the SICU and the medical units is also encouraged.

The collaboration and cooperation of the clinical laboratory in this endeavor was very important. Analyzer selection, installation, calibration, repair, and quality control testing were provided by laboratory personnel. Government regulation of laboratory analysis could impact on the recommendations of this study by prohibiting RCP management of arterial blood gas analyzers. These regulations

are currently being debated on a national level and their actual wording and impact awaits further experience.

Because this study evaluated only a small number of the ABG analyses obtained during the study periods, there is the potential for non-random error to have been introduced into the data collection. We estimate that approximately 20% of the total blood gas samples drawn during the month examined were included in the study. A possible concern in interpreting our data directly is that the criteria for appropriateness were entirely determined by retrospective chart analysis. Inadequate documentation could alter the determination of whether a blood gas was appropriate. However, we believe that this is of little concern because we have been observing this monitor for more than 5 years, and appropriateness frequency among RCPs has remained 85-95%. Even though the absolute value of appropriate frequency may be in error, the observation that improvement occurred during this study period is valid.

In conclusion, we feel that RCP-controlled ABG analysis is a method for improving appropriateness of ABG sampling. When based on a collaboratively developed and clinically useful algorithm, a marked and sustained improvement in the frequency of appropriateness of analysis can be obtained. A side benefit of this approach is improved collaborative interaction between various caregiver groups. The acceptance of such a service has been positive and has been perceived by all care providers as an improvement in the services rendered to patients in the intensive care unit. Rather than using these data in a punitive manner, we feel it is especially important that they be employed in a positive and motivational fashion.

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The High-Frequency Pneumatic Flow Interrupter: Effects of Different Ventilatory Strategies

Leandro Cordero MD, Richard Tallman PhD, Chris Reicosky BS, Phyllis Peterman RRT, and Bruce Pawel MD

We used a piglet model of respiratory failure to compare the physiologic and pathologic effects of two different ventilatory strategies provided by a high-frequency, pneumatic, flow-interrupter ventilator (HFFI). The strategies employed were continuous high frequency ventilation interrupted either by deflationary pauses or by pulsatile inflationary waves. Twelve piglets (mean age 4 days, mean birthweight 1.4 kg) randomly assigned to either the deflationary or the inflationary group received saline lung lavage and 6 hours of HFFI ventilation (10 Hz, 1.0 F_{IO_2}). Successful gas exchange was accomplished without cardiovascular compromise with both strategies. Mean airway pressures required by animals in the deflationary group were higher than those in the inflationary group. In the deflationary group, 5/6 piglets and in inflationary group 1/6 piglets presented with airway mucosal damage beyond the trachea. Significant pneumonitis was seen in all animals in the deflationary group but in only one of those in the inflationary group. In this model, the deflationary strategy led to airway and lung damage, whereas the inflationary strategy was associated with minimal airway and lung injury, perhaps due to the maintenance of high lung volume and enhanced alveolar recruitment. (Respir Care 1992;37:348-356.)

Introduction

In recent years, numerous studies of high frequency ventilation (HFV) in humans and in animals have been conducted and extensive reviews on the subject have been published.[1,2] As newer high frequency ventilators become available, it should be expected that investigators would report not only technical descriptions of the devices but also their operational characteristics and the ventilatory strategies employed.[3]

A high-frequency pneumatic flow-interrupter ventilator (HFFI)* also known as a high-frequency percussive ventilator has been used in human[4-9] and animal studies.[10-16] This device can be used for conventional mechanical ventilation (CMV) or for HFFI ventilation. It can also be programmed to deliver a combination of CMV breaths and high frequency pulses.[17]

One ventilatory strategy used in the past was a continuous oscillation interrupted by short deflationary pauses. Although successful in promoting CO_2 washout and preventing air trapping,[4,5] this strategy has been associated with significant airway injury in animals.[11,16] Another HFFI program used was a continuous high-frequency wave interrupted periodically by conventional mechanical breaths.[5,7,9] This particular strategy has the advantage of main-

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The authors have no financial interest in the products described.

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*Suppliers are identified in the Products Sources section at the end of the text.

taining lung volume while adding the benefit of periodic lung inflations. In surfactant-deficient animals, HFV strategies that maintain lung volume and promote alveolar recruitment are known to improve lung mechanics with minimal airway and lung injury.[1,18,19]

In light of the above, we designed an investigation to compare the physiologic and pathologic effects of a HFFI deflationary strategy with that of a continuous oscillatory flow interrupted by conventional tidal breaths, in a piglet model of respiratory failure.

Materials and Methods

Subjects and Preparation

Twelve newborn piglets ages 3 to 5 days with a mean (SD) age = 3.8 (4) days and birthweight of 1.1 to 1.6 kg with mean (SD) weight = 1.4 (0.25) kg were included in this study, which was reviewed and approved by the Institutional Laboratory Animal Care and Use Committee. Ten of the piglets were male and two were female. Two hours after separation from their sow, they were given 0.01 mg/kg atropine subcutaneously and ketamine/acepromazine (10:1, 20 mg/kg) intramuscularly (I.M.) and were then intubated with a triple-lumen uncuffed 3-mm endotracheal tube. Surgical preparation included cannulation of the femoral vein for placement under fluoroscopy of a 5-Fr Swan-Ganz catheter into the pulmonary artery, for mixed-venous sampling and for the continuous infusion of 5 mL · h · kg^{-1} of 10% dextrose in water ($D_{10}W$) mixed with ketamine (10 mg · h · kg^{-1}).

Heart rate and aortic and pulmonary artery blood pressures were continuously recorded, and cardiac output was determined in duplicate by thermodilution using 1 mL 5% dextrose in water (D_5W) at 26°C. The animals were placed in a servo-controlled radiant warmer with rectal and blood temperatures continuously monitored. Arterial blood gas values and hemoglobin levels were measured. Alveolar-arterial oxygen ratio was calculated from the alveolar air equation. Intrapulmonary shunts ($\dot{Q}_s/\dot{Q}_t$) were calculated with the method described by Ruiz et al.[20]

Airway pressures were measured 5 mm above the distal tip of the endotracheal tube through one of the intramural catheters while the one designed for jet ventilation remained occluded. To increase the frequency response of the system, the pressure-measuring catheter was filled with saline. The frequency response of this system is flat up to 32 Hz.[12] Pressure signals from the transducer were displayed on a multichannel recorder after calibration with a water manometer. Mean airway pressure ($\bar{P}_{aw}$)was obtained by electronically damping the pressure signal.

Ventilation

The HFFI ventilator (VDR-4) delivers gas to the patient from a pressurized source (30-65 psi, 206-448 kPa) through a pneumatic valve arrangement. Gas is interrupted repeatedly to deliver a pulsatile flow into the breathing circuit proximal to the patient's airway. Warm, humidified gas is entrained via the inspiratory circuit downstream from the auxiliary and nebulizer source gas. Flow, duration of conventional breath, and gas entrainment determine tidal volume. Periodic interruptions of the pulsatile flow can be programmed to allow return of airway pressure to near zero or to baseline PEEP/CPAP. The VDR-4 is capable of providing either conventional ventilation, high frequency ventilation, or a combination of the two.

CMV settings used in the present study were frequency, f = 18; peak inflation pressure, PIP = 20 cm H_2O, [1.96 kPa]; positive-end expiratory pressure, PEEP = 4 cm H_2O, [0.39 kPa]; and an inspiratory-to-expiratory-time ratio, I:E = 1:2 to 1:3. As the number of lavages increased, ventilator settings were adjusted to maintain arterial P_{CO_2} < 45 torr [5.99 kPa] and P_{aO_2} > 60 torr [7.99 kPa]. The average postlavage setting was f = 30, PIP = 30 cm H_2O, [2.94 kPa]. PEEP = 4 cm H_2O, [0.39 kPa]; and $\bar{P}_{aw}$ = 14 cm H_2O, [1.37 kPa].

Initial HFFI settings were f = 600; I:E = 1:1; PIP = 35 cm H_2O, [3.43 kPa]; $\bar{P}_{aw}$ = 19 cm H_2O, [1.86 kPa]; PEEP = 15 cm H_2O, [1.47 kPa]. Throughout the study, PIP and PEEP were adjusted to provide adequate gas exchange (P_{aO_2} > 60 torr [7.99 kPa], P_{CO_2} < 45 torr [5.99 kPa]). Following surgery, all animals were given 0.02 mg/kg pancuronium bromide I.V. and were placed on CMV. Lung lavage

was accomplished using 35 mL/kg warm saline (37°C) delivered and removed by gravity.[21] Ten, 1-minute lavages were performed at 5-minute intervals. This technique has been shown to decrease static and dynamic compliance and to increase intrapulmonary shunt, indicative of acute respiratory failure.[22] All animals were assigned at random (by the flip of a coin for each pair) to one of the following ventilatory strategies: the deflationary group in which the continuous oscillatory waveform was interrupted every 12 seconds by a 1-second pause, during which time the airway pressure returned to near zero, or the inflationary group in which the continuous oscillatory waveform presented a pulsatile tidal breath of 1-second duration at a frequency of 12/minute (Fig. 1). After 6 hours of high frequency ventilation, all animals were put to death with an intravenous injection of potassium chloride.

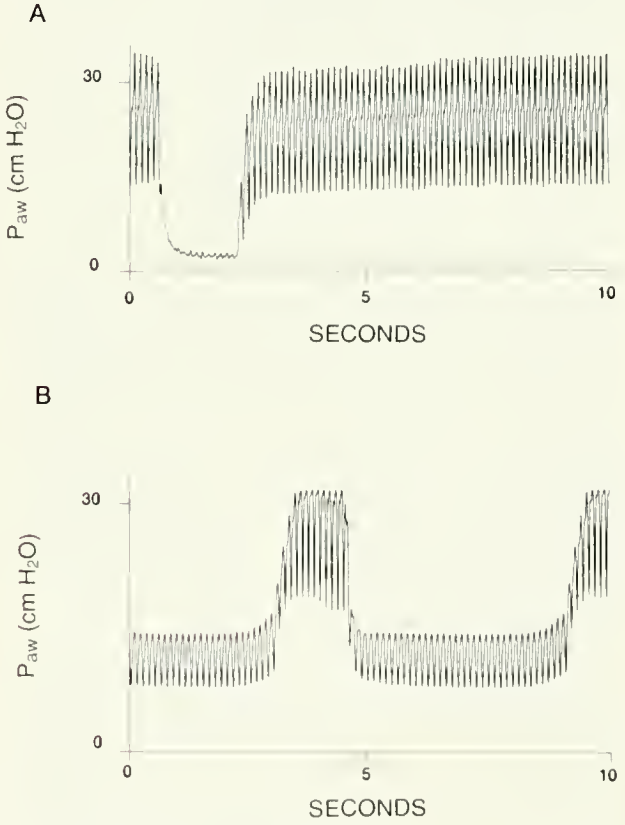

Fig. 1. High-frequency flow-interrupter waveforms: A. 1-second, 5 times/minute deflationary pause. B. 1-second, 12 times/minute pulsatile conventional tidal breath.

Pathology

At autopsy, the lungs were inflated with formalin delivered by gravity at 40 cm H_2O [3.92 kPa] pressure. The position of the endotracheal tube tip was noted at autopsy, and airway tissue cross-sections were taken from the midtrachea, carina, left and right main-stem bronchi (LMB, RMB), and left and right hilar bronchi (LH, RH). All tissue sections were formalin-fixed, paraffin-embedded, and cut at 5 μm thickness for hematoxylin-eosin staining. Two sections of parenchyma from the right lower (RLL) and left lower lung (LLL) were examined, and the presence of congestion, intra-alveolar hemorrhage, pneumonitis, bronchiovascular hemorrhage and edema was noted and scored on a severity scale of 1 to 4. The presence of necrotic material plugging bronchioles was also recorded.

The severity of the airway injury was assessed by using a scoring system that takes into account not only the type of lesion seen, but also its circumference and depth. The sum of the products of lesion circumference × lesion depth for each type of injury noted equals the airway injury score (AIS) for each airway level examined.[11] All airway sections were scored by one of the authors (BP) who was unaware of the treatment modality.

To assess differences in the severity of airway injury among the two treatment groups, mean AIS were compared using the Wilcoxon rank sum test. For the hourly derived variables, a three-way analysis of variance were used. Intergroup differences in the occurrence of pneumonitis was tested with Fisher's exact test. In all cases, a probability level of 95% ($p < 0.05$) was accepted to indicate significance. All data are reported as mean (SD).

Results

All animals in the inflationary group and 5/6 piglets in the deflationary group survived the 6 hours of HFFI ventilation. One animal in the deflationary group became irreversibly hypotensive at 4 hours postlavage. No cause of death could be determined at autopsy.

For all animals, regardless of ventilatory group, heart rate was 182 (41) beats/min following lavage and 182 (47) beats/min at the end of the experiment

Table 1. Cardiovascular and Blood Gas Values in 12 Piglets

	Lung Lavage		Hours on HFFI Ventilation		
	Before	After	First	Third	Fifth
Cardiovascular Variables					
Heart rate, beats/min	180 (31)*	182 (41)	178 (40)	178 (49)	186 (48)
Mean aortic pressure, torr	61 (8)	71 (15)	70 (14)	70 (13)	66 (17)
Cardiac index, mL/kg	0.68 (0.1)	0.58 (0.2)	0.54 (0.2)	0.52 (0.01)	0.33 (0.2)
Arterial Blood Gas Variables					
pH	7.32 (0.01)	7.30 (0.06)	7.31 (0.08)	7.34 (0.07)	7.34 (0.08)
P_{O_2}	91 (15)†	208 (177)	311 (140)	338 (187)	298 (184)
P_{CO_2}	30 (3)	35 (5)	35 (6)	36 (5)	39 (11)
$H_{CO_3^-}$	17 (2)	17 (2)	17 (2)	19 (2)	20 (2)‡

*All values are mean (SD).

†Room air, all other blood gases were on 100% O_2.

‡ HCO_3^- at fifth hour significantly different by ANOVA Repeated measures, with Tukey's multiple comparison, $p < 0.05$.

(Table 1). Mean aortic pressure was 71 (15) torr [9.46 (1.99) kPa] following lung lavage and 66 (17) torr [8.79 (2.27) kPa] 6 hours later. Cardiac index was 0.68 (0.1) $L \cdot min \cdot kg^{-1}$ at the onset and declined to about 0.33 (0.1) $L \cdot min \cdot kg^{-1}$ at the end of the study. These differences, however, were not statistically significant. Arterial blood gases were kept within the intended range (Table 1). No significant differences in cardiovascular variables or arterial blood gas values (with the exception of serum bicarbonate levels) were observed among individual animals or across the treatment groups. A mild metabolic acidosis developed over the 8-hour experiment (Table 1).

For the entire group of animals, average intrapulmonary shunt was 47% following lavage, 24% midway, and 20% at the end of the experiment. Arterial-to-alveolar oxygen ratios were similar for both treatment groups following lavage and during the first hour of HFFI ventilation (Fig. 2A). After that time and until the end of the study, P_{aO_2}-to-P_{AO_2} ratios increased noticeably for animals in the inflationary group and remained unchanged for the deflationary group. The differences in P_{aO_2}-to-P_{AO_2} ratios were statistically significant at 3, 5, and 6 hours ($p < 0.05$).

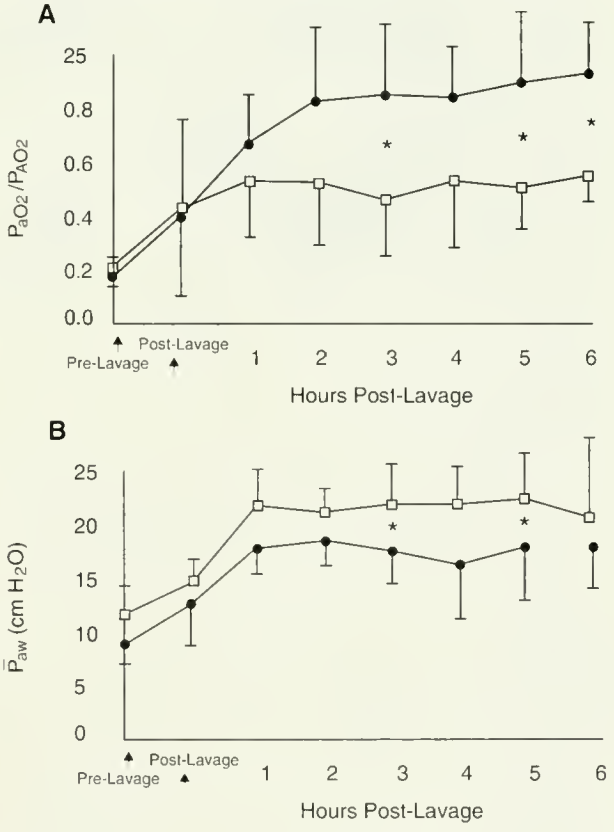

Fig. 2. Comparison of P_{aO_2}-to-P_{AO_2} ratio (A) and $\bar{P}_{aw}$ (B) between animals in the deflationary (□) and inflationary (●) groups. Mean (SD),* $p < 0.05$.

Airway Pressures

For animals in the deflationary group, PIP and PEEP averaged 33 (6) cm H_2O [3.23 (0.58) kPa] and 13 (2) cm H_2O [1.27 (0.196) kPa], respectively. For the inflationary group, PIP and PEEP averaged 34 (5) [3.33 (0.49) kPa] and 16 (2) cm H_2O [1.569 (0.196) kPa], respectively.

Following lavage and throughout the experiment, $\bar{P}_{aw}$ for animals in deflationary group were higher—20 (2) cm H_2O [1.96 (0.196) kPa]—than those in the inflationary group, 18 (3) cm H_2O [1.765 (0.29) kPa]. These differences are statistically significant ($p < 0.05$) only during the third and fifth postlavage hours (Fig. 2B). The 1-second inflationary wave increased PIP by approximately 15 cm H_2O [1.47 kPa], whereas the deflationary pause decreased the baseline to about 2 cm H_2O [0.196 kPa] (Fig. 1).

Pathology Studies

Airway mucosal lesions characteristic of necrotizing tracheobronchitis (NTB) were found in the tracheas of all animals regardless of mode of treatment. In the deflationary group 5/6 and in the inflationary group 1/6 piglets presented with significant NTB lesions beyond the trachea. Mean AIS for both groups of animals are shown in Table 2, in which it can be seen that excluding the trachea, the total mean AIS was higher for animals in the deflationary group, 7 (6), than for the animals in the inflationary group, 0.8 (1.6). These differences were statistically significant (Wilcoxon rank sum test $p < 0.05$). Furthermore, necrotic material occluding the bronchioles was found in 3 of the animals in the deflationary group, but in none of the inflationary group.

Table 2. Mean Airway Injury Scores (AIS) by Anatomic Location

	Deflationary n = 6	Inflationary n = 6
Carina	4	0.5
LMB	0.8	0.0
RMB	1.3	0.8
LH	0.5	0.0
RH	0.3	0.0
Mean (SD) AIS	7.0 (6.0)	0.8 (1.6)

* LMB = left main bronchus, RMB = right main bronchus, LH = left hilum, RH = right hilum.

Microscopic examination of the parenchyma revealed marked congestion and small alveolar hemorrhages in all animals regardless of mode of treatment. Significant pneumonitis (predominately polymorphonuclear leukocytic infiltration, Fig. 3) was seen in all animals in the deflationary group and in only 1/6 piglets from the inflationary group ($p < 0.05$ Fisher's exact test).

Discussion

Lung lavage is an accepted technique that produces clinically important surfactant depletion and predictable respiratory failure in animals.[11,21] Among many experimental choices, the piglet has become a good model not only for its availability but also for its morphologic and physiologic similarities to the human lung.[23-25] Furthermore, Davis et al,[26] studying the effects of hyperoxia and hyperventilation, concluded that piglets appear to be more prone to lung injury and exhibit a more pronounced inflammatory response than humans. Certainly, in our laboratory, lung lavage in neonatal piglets has produced a dependable model of respiratory failure.[11,14]

It is evident that, in this short-term experiment, successful gas exchange was accomplished by both ventilatory strategies without cardiovascular compromise. This observation is remarkable considering that neither strategy was designed to minimize $\bar{P}_{aw}$, which as a matter of fact was quite high for animals in the deflationary group. It is conceivable that the deflationary pauses may have intermittently promoted venous return to the heart. Also, the relatively low ventilatory frequency (10 Hz) used in this experiment may have contributed to the absence of cardiovascular side effects.[3]

Mean airway pressures needed to ventilate these animals were higher for the deflationary than for the inflationary group (Fig. 2B). This may reflect the need for maintaining a stable lung volume in the face of significant deflations or may represent absence of improvement in lung mechanics.

It is likely that the pulmonary edema and bronchoalveolar hemorrhages observed in all animals, regardless of mode of treatment, may represent nonspecific responses to lavage, hyperoxia, and mechanical ventilation. In piglets, short exposures (6-8 hours) to 100% oxygen[11,25] have not been associated with important lung injury, whereas more prolonged exposure (48 hours) resulted in marked inflammation and cell necrosis.[26]

Considering that during HFFI ventilation, expiration depends on the elastic recoil of the lung, it

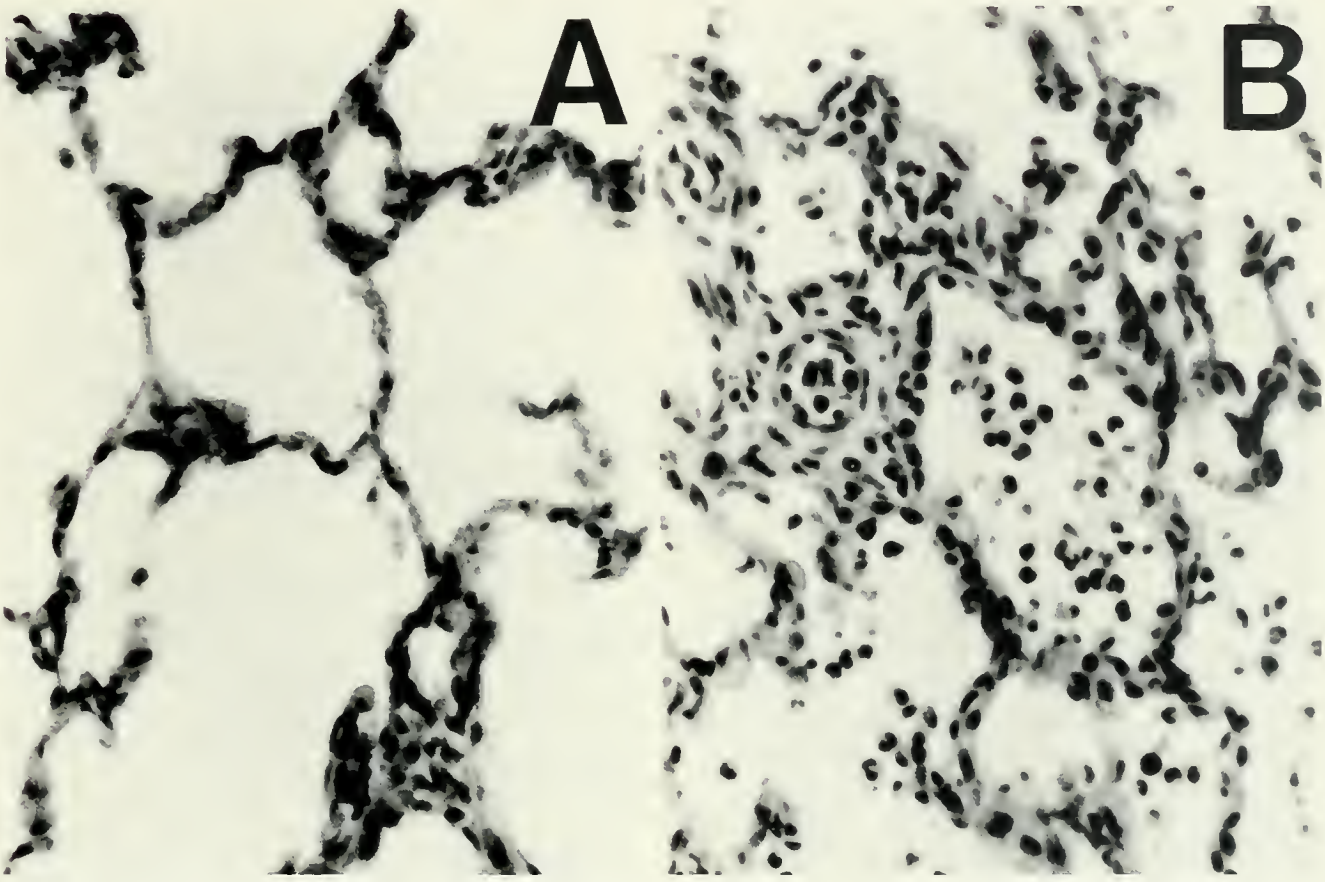

Fig. 3. A. Normal lung parenchyma in an animal in the inflationary group. B. Granulocytic infiltration seen in animals of the deflationary group (magnification × 400).

could have been expected that a strategy without deflationary pauses would increase air trapping and lead to lung rupture.[5] Perhaps because of the low frequency used (10 Hz) no such occurrences were noted.

The combination of HFFI ventilation with mechanical sighs of excessive volume and/or inappropriate timing has been reported to result in pulmonary interstitial emphysema in premature baboons, but carefully controlled sigh patterns prevent alveolar collapse without airway wall disruption.[10]

NTB has been reported to occur in humans and animals following CMV,[26,27] high frequency jet,[28,29] high frequency oscillation,[30] and HFFI ventilation.[11,15] For years, the association between NTB and HFFI was thought to be device-specific,[3] but recently it has been recognized to be related to the ventilatory strategy employed.[11,31] Humidity and temperature of the inspired gas and airway intraluminal pressure are also thought to be causally associated with NTB.[3] Which specific factor played a role in the development of NTB in the animals exposed to the deflationary strategy is unclear. Because humidity and temperature of the inspired gas and ventilator type can be excluded, it is tempting to speculate that the large intraluminal pressure swings characteristic of the deflationary strategy may have initiated the mucosal injury by altering local circulation.[33] After all, large pressure swings generated by CMV or by low-volume high-frequency oscillation have been noted to produce significant bronchoepithelial injury in surfactant-deficient rabbits.[17,18]

Pneumonitis has been observed in lungs of atelectasis-prone rabbits following CMV or low-volume high-frequency oscillation (HFO).[18,19] Due to the eosinophilic nature of these infiltrates, it has been suggested that a chemical pneumonitis has been induced by the lavage process.[18] In the present

study, the granulocytic nature of the pulmonary infiltrates and the absence of pneumonitis in 5/6 piglets in the non-deflationary group suggest otherwise. Preliminary results showed that in surfactant-depleted rabbits, CMV, unlike HFO, was associated with an intense granulocytic infiltration after only 4 hours of mechanical ventilation.[34]

Recent studies indicate that in order to be effective, high frequency ventilation must be supplemented by some type of alveolar recruitment maneuver.[1,19] It has been proposed that periodic, sustained inflations (SI) may effectively recruit alveoli if such inflations last a minimum of 10 seconds and have an amplitude of 10 cm H_2O above baseline.[35] Watanabe et al,[36] on the other hand, has reported that in surfactant-depleted rabbits, adequate lung volume can be maintained with repeat SI of only 1-5 seconds duration and 25-30 cm H_2O pressure. It is tempting to speculate that the conventional breath used in the present investigation may be comparable to a 1-second SI. The pulsatile nature of this tidal breath could have only added further benefit to this ventilatory strategy, if, as proposed by Byford et al,[37] pulsatile SI increased lung volume more than static SI. In the absence of direct lung-volume measurements, we can only speculate that the continuous oscillatory wave may have maintained high lung volume and that the 1-second inflation could have recruited more alveolar units. The paucity of airway damage and the absence of significant lung injury in the inflationary group could be explained by the fact that strategies that encourage alveolar expansion and high lung volume maintenance result in improved pulmonary mechanics and diminished anatomic damage.[18,19,38] The fact that P_{aO_2}-to-P_{AO_2} ratios for animals in the inflationary group increased over time (Fig. 2A) may reflect an improvement in pulmonary mechanics or a successful prevention of additional injury.[38]

In summary, a deflationary strategy in this piglet model of respiratory failure leads to airway and pulmonary granulocyte-mediated injury. The inflationary strategy seems to be associated with either absent or modest airway and lung injury. It is likely that the pulsatile conventional breath enhances alveolar recruitment, whereas HFFI ventilation successfully maintains lung volume.

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PRODUCT SOURCES

HFFI Ventilator:
VDR-4, Bird Space Technology, Sand Point ID

Triple-Lumen Endotracheal Tube:
NCC, Mallinckrodt, St Louis MO

Thermodilution Calculator:
Am Edwards 9520A, Baxter Healthcare, Irvine CA

Radiant Warmer:
Air Shields, Model DR, Air-Shields Vickers, Hatsboro PA

Blood Gas Analyzer:
Radiometer ABL 2 RA, Radiometer America Inc, Westlake OH

Pressure Transducer:
Gould P23 ID, Gould Inc, Cleveland OH

Heated Humdifier:
Conchatherm III, Respiratory Care Inc, Arlington Heights IL

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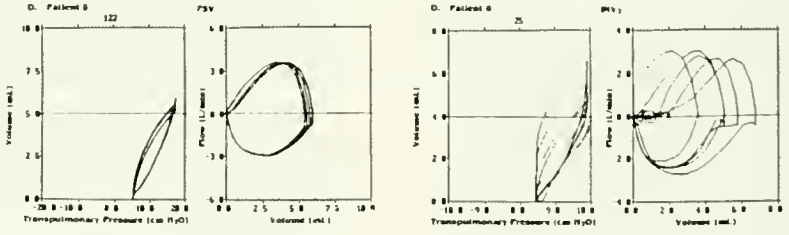
CORRECTIONS

We regret the omission of Rex Marley MS CRNA RRT from the Reviewer Appreciation List that appeared in the December 1991 issue.

Also, two errors appear in the paper "Feasibility of Applying Flow-Synchronized Ventilation to Very Low Birthweight Infants" (Respir Care 1992;37: 249-253).

The p value for the difference between $\dot{V}_E$ during IMV_1 is significantly different from the value during IMV_2 and should be 0.016 (not 0.143 as shown in Table 2).

In Part D of Figure 1 the flow-volume and pressure-volume loops on the left should be labeled IMV_1 and the flow-volume and pressure-volume loops on the right should be labeled FSV. The correctly labeled figure is shown below.



We regret the errors.

The Effect of Oral versus Nasal Breathing on Oxygen Concentrations Received from Nasal Cannulas

Crystal L Dunlevy EdD RRT and Sylvia E Tyl BS RRT

BACKGROUND: A review of the literature reveals that research on the effects of mouth vs nose breathing and resting (or 'normal') vs hyperventilation on oxygen concentrations delivered by nasal cannula has been limited. MATERIALS & METHODS: 15 healthy subjects fitted with hypopharyngeal catheters for gas sampling and nasal cannulas at 2 L/min breathed at resting and hyperventilation levels through valved systems that allowed only mouth breathing or only nose breathing. RESULTS: Oxygen concentrations were significantly different with mouth-open vs mouth-closed at resting and hyperventilation levels and with mouth-closed resting vs mouth-closed hyperventilation. Mean F_{IO_2} with mouth-open resting ventilation at 2 L/min was only 0.24 and with mouth-open hyperventilation only 0.23. CONCLUSION: The data suggest that guidelines for the use of the nasal cannula and adjustment of flowrates may need to be re-examined. Breathing through the nose should be encouraged for maximum F_{IO_2} at a given oxygen flowrate. (Respir Care 1992;37:357-360.)

Background

An understanding of oxygen therapy is essential to the delivery of effective respiratory care. The nasal cannula is commonly used for oxygen delivery, yet the major respiratory care textbooks differ in the information that they provide on the therapeutic ranges and limitations of the device.[1-4] Because of the widespread use of nasal cannulas in both hospital and home care settings and the inconsistencies found in respiratory care textbooks, we undertook a study of the effects of oral versus nasal breathing at two levels of ventilation on oxygen concentrations received from cannulas.

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A version of this paper was presented by Dr Dunlevy during the RESPIRATORY CARE OPEN FORUM at the 1991 AARC Annual Meeting in Atlanta, Georgia.

Reprints: Crystal Dunlevy EdD RRT, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, 1583 Perry St, Columbus OH 43210-1234.

Our review of the literature revealed limited research on the use of the nasal cannula.[5-7] Two of those studies examined the effects of oral versus nasal breathing on oxygen concentrations received. Kory et al[5] included five subjects and reported no significant variation in oxygen concentrations received by mouth-open and mouth-closed breathing. Subjects were instructed to breathe "normally," but no respiratory rates or tidal volumes were reported with the data. Conversely, Poulton et al[6] reported a significant difference in oxygen concentrations received with oral versus nasal breathing. Their study reported results on the basis of only one subject. Redding et al[7] investigated oxygen concentrations received from commonly used delivery systems, including the nasal cannula, but did not compare oral and nasal breathing or normal ventilation versus hyperventilation.

Materials and Methods

Fifteen healthy subjects between the ages of 20 and 25, with no history of pulmonary or cardiac disease and no history of smoking, participated in the study. The naso- and hypopharynx of each subject was anesthetized with 10% xylocaine. Under

direct vision, a 6.5-French suction catheter was inserted through the nose into the hypopharynx and secured. Subjects were then fitted with a nasal cannula at 2 L/min. Flowrates were verified with a flowrater.* Subjects were instructed to vary their breathing pattern, alternating between mouth-open resting-level ventilation; mouth-open hyperventilation; mouth-closed resting-level ventilation; and mouth-closed hyperventilation. The order of breathing patterns was randomly determined by having each participant draw from a container containing slips of paper describing the four patterns. Subjects breathed through a mouthpiece with one one-way valve for mouth-closed breathing (Fig. 1A) and two one-way valves for mouth-open breathing (Fig. 1B). (During mouth-closed breathing, the subject inhaled through the nose and exhaled through the mouthpiece, whereas during mouth-open breathing the subject inhaled and exhaled through the mouthpiece.) Respiratory rate was monitored and controlled by having subjects breathe with a metronome at a rate of either 14 or 24 breaths per minute. Subjects maintained their normal tidal volume (400-700 mL [0.04-0.07 L]) throughout the study (verified with a respirometer, Figs. 1). End-tidal carbon dioxide partial pressures (P_{etCO_2}) measured via a calibrated end-tidal CO_2 monitor were used to verify the presence of normal ventilation and hyperventilation. Normal P_{etCO_2} values were defined as 35-45 torr [4.67-599 kPa]; hyperventilation was indicated by P_{etCO_2} values ≤ 34 torr [4.53 kPa].

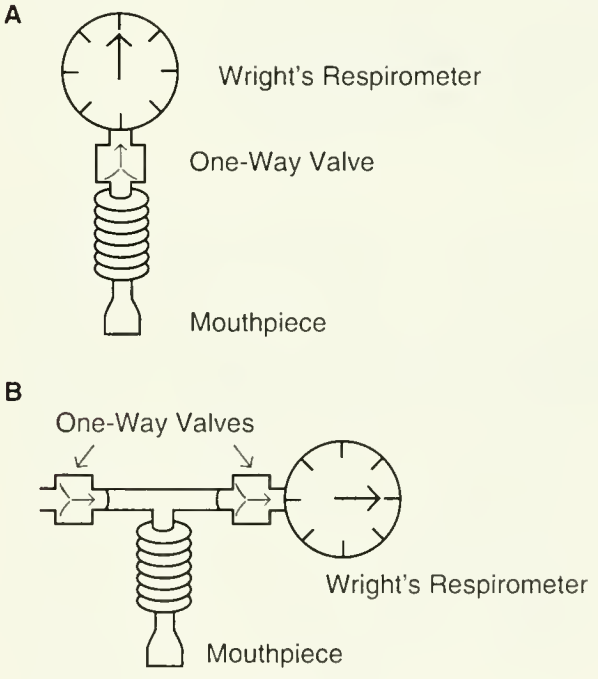

Fig. 1A. Apparatus used for breathing with the mouth closed. Fig. 1B. Apparatus used for breathing with the mouth open.

*Suppliers are identified in the Product Sources section at the end of the text.

After subjects had maintained a pattern for 5 minutes, hypopharyngeal gas samples were aspirated using a glass syringe with a 3-way stopcock. Gas samples were analyzed for P_{O_2} by a calibrated blood gas analyzer.[8] F_{IO_2} values were calculated as follows:

$$F_{IO_2} = \frac{P_{O_2} \text{ of gas sample}}{P_B - 47},$$

where P_B is the measured barometric pressure.

Means and standard deviations were calculated for each set of F_{IO_2} and P_{etCO_2} values. Two-tailed, paired *t* tests were applied, with $p < 0.01$ considered statistically significant.[9]

Results

The mean F_{IO_2} values at each breathing pattern (with standard deviations) are listed in Table 1. Results of statistical analysis comparing F_{IO_2} values obtained during the various breathing patterns are shown in Table 2. All comparisons yielded statistically significant differences except mouth-open resting-level vs mouth-open hyperventilation. Table 3 contains results of comparison of P_{etCO_2} for each breathing pattern.

Table 1. Delivered Oxygen Concentrations at Various Breathing Patterns in 15 Normal Subjects

Breathing Pattern	F_{IO_2} Mean	F_{IO_2} SD
Mouth-open resting-level ventilation	0.24	0.03
Mouth-open hyperventilation	0.23	0.03
Mouth-closed resting-level ventilation	0.31	0.06
Mouth-closed hyperventilation	0.27	0.03

Initially, three sets of data were invalidated because of extremely high P_{O_2} values obtained

Table 2. Comparison of Delivered Oxygen Concentrations with Mouth-Open and Mouth-Closed Breathing Patterns at 2 Levels of Ventilation in 15 Normal Subjects

Breathing Pattern	t	p
Mouth-open vs mouth-closed resting-level ventilation	6.24	0.00001
Mouth-open vs mouth-closed hyperventilation	5.28	0.0001
Mouth-closed resting-level ventilation vs hyperventilation	3.96	0.0001
Mouth-open resting-level ventilation vs hyperventilation	1.47	0.16

from the gas samples. The problem was believed to be caused by partial obstruction of the hypopharyngeal catheter and/or the nares with the xylocaine jelly that was used to anesthetize the nares. As a consequence, we discontinued use of the xylocaine jelly and anesthetized the nares with xylocaine spray, thereby eliminating the problem.

Discussion

Cannulas are low-flow devices and the factors (primarily inspiratory flowrate) that affect oxygen concentration received from such devices are well known. However, authors have suggested that mouth vs nose breathing (at least at resting levels) has little effect on concentration received from nasal cannulas, perhaps in part because of the reservoir provided by the upper airway.[4] Our results suggest that oxygen concentration received during mouth breathing is significantly lower than oxygen concentration received during nose breathing both during resting-level- and hyperventilation and that hyperventilation significantly lowers oxygen concentration received during mouth-closed resting-level ventilation compared to oxygen concentration received during mouth-closed hyperventilation.

Table 3. Comparison between P_{etCO_2} Values Obtained with Mouth-Open Resting-Level-ventilation and Hyperventilation and Mouth-Closed Resting-Level Ventilation and Hyperventilation in 15 Normal Subjects

Breathing Pattern	t	p
Mouth-open resting-level ventilation vs hyperventilation	5.17	0.0001
Mouth-closed resting-level ventilation vs hyperventilation	4.98	0.0002

A limitation of our study is that it was conducted on 'normal' subjects; therefore, the conclusions drawn may have limited application to other populations, such as those with cardiac or pulmonary disease. The data suggest that guidelines for the use of the nasal cannula may need to be re-examined.

Results of this study suggest that while breathing through the mouth, subjects received little more than room air F_{IO_2} (0.24 and 0.23 during resting-level- and hyperventilation, respectively). However, the F_{IO_2} increased significantly when subjects breathed through the nose, and the increase of 4-8% was clinically important. This may explain why COPD patients often place their cannula in their mouth during periods of exercise or dyspnea. It may be beneficial to instruct patients who are receiving oxygen via nasal cannula to make an effort to breathe through the nose. If patients cannot breathe primarily through the nose, it may be necessary to consider alternate methods of oxygen delivery in order to increase both the therapeutic efficacy and cost-effectiveness of the therapy.

Further study should be undertaken to investigate the therapeutic ranges of other oxygen delivery devices at various flowrates. Because the results of this study are supported by Poulton et al,[6] and refuted by Kory et al,[5] further investigation is also indicated with regard to the nasal cannula's therapeutic guidelines.

ACKNOWLEDGMENTS

We thank the members of the 1992 Respiratory Therapy Class at Ohio State University for being cannulated!

PRODUCT SOURCES

Flowrater:
Precision Bore Flowrater Model #10A 1755, Fischer & Porter Co, Warminster PA

Respirometer:
Wright's respirometer, Ferraris Medical Inc, Holland NY

Arterial Blood Gas Analyzer:
Corning 165 Blood Gas Analyzer Corning Medical, Corning Glass Works, Medfield MA

End Tidal CO_2 Monitor:
Nellcor End-Tidal CO_2 Monitor, Nellcor Inc, Hayward CA

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The Bronchodilator Properties of Magnesium Sulfate

Hugh S Mathewson MD

On the Epsom Common, in 1618, an English farmer found a water hole, but his cattle would not drink the water. When a sample of the water was evaporated to dryness, it was found that the water contained a high concentration of "Epsom salts," later to be identified as magnesium sulfate. Its use as a medicinal water was suggested by Lord Dudley North in 1645, and its value as a purgative soon became well known.[1] Late in the 19th century, solutions of magnesium sulfate were given intravenously, and the toxic syndrome of hypermagnesemia was described, which included profound muscular relaxation, sedation, and depression of cardiac conduction.[2] Parenteral injections of the drug began to be used to counteract seizure states associated with tetanus, nephritis, and eclampsia. Later, obstetricians found magnesium sulfate to be useful in controlling uterine hypertonus.

In 1937, French investigators showed that magnesium salts protect animals against anaphylactic shock.[3,4] From this observation Haury[5] postulated in 1940 that asthmatic patients might be hypomagnesemic and that administration of magnesium would be helpful in alleviating bronchospasm. He found that about half of a series of asthmatic patients had subnormal serum magnesium levels, but the deficiencies were usually mild. He reported two cases of successful treatment of severe asthma with combined intravenous and intramuscular magnesium sulfate. A similar experience had been recorded in Argentina in 1936.[6]

From these early studies, the depressant effects of magnesium ion on the contractility of all types of muscle were revealed. Further, it was noted that calcium ion restores the tone of magnesium-depressed muscle. Eventually, the central function of calcium ion in all contractile processes was recognized, and the mutually antagonistic actions of calcium and magnesium became apparent.[7] It is now recommended that whenever magnesium sulfate is given parenterally, a solution of a calcium salt be immediately available as an antidote.[8] The development of calcium channel-blocking drugs for treatment of cardiovascular conditions focused attention on the essential role of calcium ion in regulating contractility of the vasculature. Calcium-channel blockers were also shown to inhibit the contractility of bronchial smooth muscle and to exert a protective effect against histamine-induced bronchoconstriction in asthmatic subjects.[9-11] No drugs belonging to this class have been advanced for clinical use as bronchodilators, principally because of their vasodepressor effects. However, the finding has triggered a renewed interest in the use of magnesium salts as calcium ion antagonists in bronchial smooth muscle.[12]

Okayama and colleagues[13] studied the bronchodilator effect of magnesium sulfate infusions in 10 asthmatic patients with mild attacks. Significant increases in forced vital capacity (FVC) and forced expiratory volume (FEV) and decreases in respiratory resistance (R_{aw}) were recorded, but these actions were transitory. They concluded that the rapid bronchodilatation produced could be useful in the emergency treatment of severe asthma. Two subsequent studies have supported this opinion.[14,15] Both focused upon patients treated for acute episodes in the emergency department.

The first of these studies[14] identified "poor responders" to conventional nebulized beta-agonist therapy. Of the 38 adult patients so identified, 19 received 1.2 g of magnesium sulfate by infusion, the other 19 were given placebo. The increases in peak expiratory flow were only moderately higher in the treated group, but of the 19 treated patients only 4 were admitted to the hospital, compared to 12 in the placebo group. The second report[15] involved 12 patients with severe asthma, of whom 10 benefited from intravenous magnesium sulfate in total doses of 3 g. However, subsequent albuterol inhalations were given to these patients, which resulted in significantly greater improvement. The doses of magnesium in both studies were considerably below the toxic threshold, and no systemic side actions beyond transient flushing or mild sedation were noted.

The Okayama group recently reported two cases of status asthmaticus requiring intubation and mechanical ventilation, which were successfully treated with magnesium

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sulfate infusions of 0.5 mmol/min.[16] Spirometric indices were improved and "piping" rales were diminished. The investigators suggested that the immediate, though evanescent, effect of magnesium ion could be of great benefit pending the definitive action of parenteral corticosteroids.

The foregoing clinical reports indicate that intravenous magnesium sulfate is a rapidly effective bronchodilator. It does not affect central respiratory drive, and the only potential hazard of ventilatory depression is from neuromuscular blockade. This can be anticipated by periodic checks of deep tendon reflexes. Magnesium also inhibits catecholamine-induced arrhythmias, a property that could increase the safety of beta-agonists in patients with irritable hearts.[17] Dosage criteria are still indefinite; although magnesium is efficiently handled by the normal kidney, it could be hazardous in patients with renal insufficiency. The recent interest in magnesium therapy is promising, but the indications and dosage regimens have yet to be established.

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By Robert Chatburn, RRT. Knob-turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation.

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Abnormal Radiograph in a Patient with a History of Lung Cancer, Radiotherapy, and Working in a Quarry

Digpal Chauhan MD and John Brandsburg PhD

A 67-year-old man was admitted to the hospital with the chief complaints of a 12-to-14-pound weight loss, malaise, chronic cough with expectoration of grayish sputum, and exertional dyspnea. He had not experienced fever, chills, or constitutional symptoms and had no history of pleuritic chest pain, hemoptysis, or wheezy dyspnea.

In 1972, percutaneous needle aspiration of a mass in the superior sulcus of the apex of the left lung had revealed an undifferentiated lung cancer. He received 5,000 rads of radiotherapy, and subsequent chest radiographs showed complete resolution of the mass. The patient's response to tuberculosis testing (PPD, purified protein derivative) had been negative in the past, with no data available on candida control. He had no history of bronchial asthma. The patient, who had lived in Kansas and Missouri all of his life, had been employed crushing rock in a quarry for 10 years and also had worked with heavy machinery and welding. He had a 70-pack-year smoking history.

Physical examination revealed a well-developed man in no distress. He weighed 154 lb, was 6 ft 1 in tall (ideal body weight 186 lb), was afebrile, and gave no evidence of adenopathy or clubbing. Percussion and auscultation of the chest revealed dullness in both upper lung fields and a few crackles in the left anterior chest. No wheezes or rubs were detected.

Laboratory analysis revealed normal values for hematocrit, white blood count, serum creatinine, and liver function tests. The serum IgE level was normal. P_{aO_2} was 74 torr on room air. Spirometry showed a forced expiratory volume in the first second (FEV_1) of 2.99 L and forced vital capacity (FVC) of 4.40 L, with FEV/FVC% of 68. FEV_1 did not increase with bronchodilator administration. The chest radiograph taken on admission is shown in Figure 1.

Fig. 1. Posteroanterior chest film of patient with history of lung cancer, radiotherapy, and occupational exposure to rock dust.

Questions

Radiographic Findings: What abnormalities are seen in Figure 1?
Diagnoses: What are the possible diagnoses?
Further Tests: What further tests are indicated?
Diagnosis and Therapy: What is the diagnosis and what therapy is indicated?

Answers and Discussion on Next Page

Dr Chauhan is Chief of Pulmonary Medicine and Dr Brandsburg is Microbiologist, Dwight D Eisenhower Veterans Administration Hospital, Leavenworth, Kansas.

Answers and Discussion

Radiographic Findings: Bilateral upper-lobe infiltrates are evident, with some cystic changes in the right-upper lobe.

Possible Diagnoses: With this man's history, the possible diagnoses include opportunistic indolent infections, inorganic dust fibrosis or pneumoconiosis (eg, silicosis, silicosiderosis), radiation fibrosis, and allergic bronchopulmonary aspergillosis. Possible indolent infections include pulmonary mycobacterial and fungal infections, particularly chronic pulmonary histoplasmosis and chronic necrotizing aspergillosis. Other possible diagnoses include chemotherapy-induced pulmonary fibrosis, upper-lobe fibrosis due to ankylosing spondylitis, atypical sarcoidosis, and organic dust (hypersensitivity) pneumonitis. Both silicosis and *M tuberculosis* infection are common in hosts with occupational exposure to quarry activities (silico-tuberculosis).

Further Tests: Bronchoscopy with upper-lobe biopsies and bronchial washings and brushings for inspection and cultures for acid-fast bacilli and fungi, and serologic tests are indicated.

Diagnosis and Therapy: Left-upper-lobe biopsies were negative for cancer cells and silicotic nodules. Bronchial washings and brushings and tissue cultures from the left upper lobe grew *Histoplasma capsulatum*. The patient was initially treated with 400 mg ketoconazole; however, following relapse on ketoconazole he was treated with intravenous Amphotericin B. Figure 2 is a chest film made shortly before discharge.

Fig. 2. Posteroanterior chest film of patient in Figure 1 after treatment with ketoconazole and intravenous Amphotericin B.

Discussion

Although infection with *H capsulatum* may be virtually universal in endemic areas, most cases are asymptomatic and of no clinical consequence.[1] Chronic pulmonary histoplasmosis was first diagnosed in living patients in 1948 among sanitarium patients in whom it had been mistaken for pulmonary tuberculosis, which it closely mimics.[1]

Epidemiology

H capsulatum, the causal agent, of histoplasmosis is a dimorphic fungus with its mycelial phase in the enviroment and its yeast phase at body temperature in tissues.[1,2] The fungus is ubiquitous in soil and has been found in more than 50 countries in temperate climates worldwide. It is more common in rural areas, although large urban outbreaks of histoplasmosis have been reported.[3] The fungus is found in bird and bat droppings and in high concentrations in chicken coops and starling roosts. Birds may carry the fungus in their feathers without it affecting them. It is endemic in the eastern part of the central United States including Missouri, Indiana, Kentucky, Mississippi, Tennessee, Illinois, and the Ohio River Valley.[1,2] Most cases are sporadic from casual exposure to environmental spores.[2]

Pathogenesis of Infection

Chronic histoplasmosis occurs as the direct result of primary infection in an abnormal lung and is not the result of endogenous reactivation. The fungus grows as a fluffy white mycelium that bears microconidia and tuberculate microconidia, and infection occurs by inhalation of microconidia, or spores.[1,2]

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Pathology

Histoplasmosis usually affects apicoposterior segments of upper lobes. The early lesion is an intense interstitial pneumonitis resulting from dispersion of antigen.[2] The pneumonitis is usually segmental but may be sub- or multisegmental. Focal areas of infarct-like necrosis develop in the pneumonic process, with single or multiple cavitations. The cavitary lesions of upper lung fields are usually bilateral. Cavities may be thin-walled (≤ 2 mm) or thick-walled (> 2 mm). Thin-walled cavities may represent bullae, and persistent infection is unusual in them. Roughly 80% of thick-walled cavities may be persistently infected and may progress to "marching" cavities, with progressive destruction of contiguous lung tissue.[2] Bronchogenic dispersion or aspiration of antigenic material to dependent areas can give rise to basal pulmonary fibrosis preceded by intense 'spillover' interstitial pneumonia.

Risk Factors

Chronic pulmonary histoplasmosis occurs most commonly in patients with centrilobular or bullous emphysema but may also be associated with an underlying immunosuppressive disorder.[3] Spirometry may show mild-to-severe obstruction to airflow.[2] Age, male gender, and white race are other risk factors. The classic patient is a white man of middle-age or older with a positive smoking history.

Clinical Features

Chronic cough (with or without sputum production) is common.[1,2] Hemoptysis is rare. Other symptoms include malaise, easy fatigability, weight loss, aching chest pains, low-grade fever, night sweats, and dyspnea. Laboratory analysis may reveal anemia, lymphopenia, elevated alkaline phosphatase, and increased T-helper-to-T-suppressor lymphocyte ratio.[3]

Radiology

Chest radiographs show reticular or segmental infiltrates with multiple cavitations in the apicoposterior segments of one or both upper lobes.[2] Basal interstitial pneumonia or fibrosis may be seen.

Diagnosis

Diagnosis is made by cultures, special stains, and serology.[4] Serology includes complement fixation test (CF), immunodiffusion (ID), and new radio-immunoassay (RIA).[5,6] CF is a good screening test and has a sensitivity of about 90%. New RIA detects both IgG and IgM against a histoplasmin extract and has a sensitivity of about 95%.[6] ID is more specific and less sensitive than CF.[5] A positive M band by ID is fairly specific and indicates active disease. Positive ID for both M and H bands in the same serum is highly specific for current infection. A single CF titer of 1:64 or above is fairly diagnostic as is a four-fold rise in titer of current histoplasmosis. A CF titer between 1:8 and 1:32 suggests histoplasmosis.[5]

Firm diagnosis depends on demonstration of the fungus in sputum, bronchial washings, or tissue cultures,[1,2] although cultures may be negative in patients with asymptomatic (so-called self-limiting) histoplasmosis.[5] Culture recovery from sputum is difficult and more than six sputum cultures may be required. Morphologically compatible organisms on Wright stain of sputum or silver methenamine tissue stain are usually acceptable microbiologic evidence of histoplasmosis.[2]

Chronic pulmonary histoplasmosis is easier to diagnose by serology and sputum culture than are acute and disseminated histoplasmosis. Therapy of chronic pulmonary histoplasmosis depends on the severity of illness. Ketoconazole is effective for mild-to-moderate illness and should be the initial therapy for all patients with nonmeningeal disease who are not critically ill.[7,8] In our experience, Amphotericin B should be reserved for severe life-threatening illness and relapse cases.

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Treatments for Asthma and Consumption in 1903

ASTHMA: ***Symptoms***—A painful difficulty of breathing, recurring at intervals, with a sense of tightness across the breast; a wheezing cough, hard at first, but towards the end of each paroxysm more free, and followed by the discharge of a little mucus. The attacks of asthma are generally in the nighttime, though they sometimes come on in the course of the day; and at whatever time they come on it is for the most part suddenly, with a sense of tightness across the breast, impeding respiration. The person, if in bed, is obliged immediately to get up, and he requires the free admission of air. The difficulty of breathing increases, and is performed slowly, and with a wheezing noise. These symptoms sometimes continue for hours together; and a remission takes place by degrees; the breathing becomes less laborious, and the patient speaks and coughs more easily; and if there is something expectorated, the remission is greater and sleep comes on. In the morning, and through the day, though the breathing is better than during the fit, it is not yet free from difficulty; a degree of tightness is still felt, and a very slight motion of the body is apt to bring back the paroxysm. In the evening, the breathing is worse, and about the same hour as on the former night, generally between midnight and two o'clock, the same train of symptoms is renewed. After the fits have recurred for several nights in this manner, they suffer more considerable remissions; and for some time, asthmatics may be free from complaint; but through the whole life the paroxysms are ready to return, though in different circumstances in different patients.

Asthma seldom appears before the age of puberty, and seems to attack men more frequently than women; and in persons of a full habit whom it continues to attack, it commonly causes a great degree of emaciation. Though it does not often destroy life in the paroxysms, it may become fatal by passing into other diseases, as into consumption of the lungs, or by occasioning dropsy, and many cases which have appeared a common spasmodic asthma, have been found at last to depend on organic diseases of the heart and great vessels.

Causes—Some have the fits of spasmodic asthma brought on by heat, whether of the weather or of warm apartments; and frequently by warm bathing. Some are hurt by cold and moist air, or by anything worn tight about the breast, or by distension of the stomach from a full meal, or windy diet; or from exercise hurrying the circulation of the blood. Sometimes the disease is brought on by causes affecting the nervous system, as passions of the mind; or by particular smells, or irritations of the lungs from smoke or dust.

Remedies—***Quick Relief***—Although a lobelia, or some other emetic, has for a long time been considered the only hope for relief, yet, more recently, the inhalation of chloroform has proved generally a much quicker relaxant, and consequently, the more satisfactory remedy. It is not necessary to breathe it to entire unconsciousness, but simply to relieve by putting a bottle of it—an ounce is sufficient to buy at a time—first to one nostril, closing the other with the thumb of the opposite hand, and, the mouth being closed, draw in a long and deep breath to the fullest extent the lungs will allow; then alternate with the other nostril in the same way until you realize the needed relief, or to the number of two or three times to each

This material is from "Dr. Chase's Third, Last, and Complete Receipt Book and Household Physician," by Dr Albin Wood Chase, which was published in 1903 by Crescent Publishing Co, Minneapolis, Minnesota. In a section entitled "In Memorium," at the beginning of the book, the publisher writes about Dr Chase that "His desire for knowledge was so strong, coupled with an ambition equally peculiar to his naturally energetic disposition, that he far outstripped his more dilatory companions of all institutes of learning."

These excerpts from Dr Chase's book were made available to RESPIRATORY CARE by Crystal L Dunlevy EdD RRT, Assistant Professor and Director of Clinical Education, Respiratory Therapy Division, School of Allied Medical Professions, The Ohio State University, Columbus, Ohio.

nostril. Then if not relieved, wait a few minutes and do the same again. It is better thus than to continue until unconscious. The chloroform is very satisfactorily inhaled from a glass tube inhaler, to be corked up when not in use.

Remedies—*Relief In*—A friend of mine who had had asthma, so that, at one time, he did not go to bed for five years, but took his sleep in a rocking chair, has found great relief inhaling nitrated stramonium. He says, "I gather the green leaves of the stramonium, after the plant blossoms, and dry them in the shade. When dry, I soak them a few hours in a strong solution of purified nitre, 3 ounces to soft water, 1 pint. Powder the nitre finely, and pouring on the water hot, quickly dissolves it. Soak the previously dried leaves in this solution, re-dry in the shade, then pulverize the leaves and keep from the air in box or bottle. ***To Use*—**Put a rounding teaspoonful of the nitrated powder on a plate, and touch a lighted match to the heap, when, if properly done with the purified nitre, it burns without a blaze, throwing off considerable smoke. Place a small funnel over it, and breathe the smoke arising from it by holding the mouth as close to the funnel as possible, to inhale as much as you can of the fumes. It will cause some coughing at first, but this helps to clear the throat and bronchial tubes of phlegm and soon subsides and gives very great relief."

Some persons think that sage, belladonna, and digitalis, the dried leaves of each, with the dry stramonium, all in equal proportions, nitrated, as above, and inhaled in the same manner, is preferable to the stramonium alone. If I were to use them, however, I would not use more than half as much of the belladonna and digitalis as I did of the sage and stramonium.

Whenever the inhalation of chloroform, or nitrated stramonium, etc., above given, fails, then 20 to 40 drops of laudanum, according to robustness of the patient, or the severity of the case, with 15 to 30 drops of sulfuric ether, put into a glass with a little water, and immediately drank, will almost always give relief at once. This should not be taken often enough to establish the habit of opium eating, which would prove a disease in itself, as bad as asthma, and as difficult to cure.

Remarks—In closing the subject of asthma, I would say in addition only, that according to the condition of the system, any existing difficulty, as costiveness, liver or kidney complaint, must be met and overcome on general principles, that is, to treat them as you would if they existed alone. Do all, as per instructions given under each head referred to, and very many cases of asthma will be cured, the general opinion to the contrary, notwithstanding. The condition of the surface, to keep it clean and the blood freely circulating therein, by salt washings, dry rubbings, etc, must not, in any case, be neglected in any long standing disease. If neglected, it is at your own peril.

CONSUMPTION: This disease is probably the greatest existing scourge of the human race, at least in the northern and middle latitudes. It is not deviating far from the truth to say that it causes about one-sixth or one-seventh of all the deaths north of the tropics. The duration of the disease is exceedingly variable. While some cases run their course to a fatal termination in less than a month, others have been known to continue thirty or forty years. The greater number of cases, as a rule, terminate in from one to two years. It is pre-eminently a hereditary disease.

Symptoms—The earliest symptom of consumption that usually manifests itself is a short, dry cough, exciting no particular attention, being attributed to a slight cold. It, however, continues, and after a time, increases in frequency. The breathing is more easily hurried by bodily motion, and the pulse becomes more frequent, particularly after meals and towards evening. Towards evening, there is also frequently experienced a slight degree of chilliness, followed by heat and nocturnal perspirations. The patient becomes languid and indolent, and gradually loses strength. After a time, the cough becomes more frequent, and is particularly troublesome during the night, accompanied by an expectoration of a clear, frothy substance, which afterwards becomes more copious, viscid and opaque, and is most considerable in the morning; the sputa are often tinged with blood; or haemoptysis occurs in a more marked form, and to a greater extent. As the disease advances, the breathing and pulse become more hurried; the fever is greater, and the perspirations, more regular and profuse. The emaciation and weakness go on increasing, a pain is felt in some part of the thorax, which is increased by coughing, and

sometimes becomes so acute as to prevent the patient from lying on the affected side. All the symptoms increase towards evening; the face is flushed; the palms of the hand and soles of the feet are affected with a burning heat; the feet and ankles begin to swell, and in the last stage of consumption, there is nearly always profuse diarrhoea. The emaciation is extreme; the countenance assumes a cadaverous appearance, the cheeks are prominent, the eyes, hollow and languid. Usually the appetite remains entire to the end, and the patient flatters himself with the hope of a speedy recovery, often vainly forming distant projects of interest or amusement, when death puts a period to his existence. Tubercular deposits are also usually found in other organs of the body; the liver is enlarged and changes in appearance, and ulcerations occur in the intestines, the larynx, and trachea. These are so frequent and uniform as to lead to the belief that they form part of the disease.

Causes—The causes of this disease are divided into remote and exciting. Of the former, the most important is hereditary predisposition. It is not, however, an actual cause of the disease; and hence, there are many cases in which the children of consumptive parents do not fall a prey to this disease; but it renders those who are in that condition much more liable to be affected by the exciting causes. Whatever weakens the strength of the system, or interferes with the oxygenation of the blood, tends to the production of the disease. Hence living in bad air, insufficient and unwholesome food, and sedentary pursuits, tend to it. Among the more exciting causes are exposure to cold or damp, especially after the body has been heated, intemperance of any kind, profuse evacuations, and exposure to the reception of dust into the lungs, as in the case of certain artificers, needle-pointers, stonecutters, and the like.

Remedies—As prevention is better than cure for those who are liable to have consumption, but have not got it fastened upon them yet, I will give the rules of the celebrated Dr. S.S. Fitch, of New York, for its prevention, as they are certainly valuable and ought to be heeded by everyone. He claims an absolute preventive in all cases and all persons, but as his rules are so very strict, if they are lived up to, they will certainly do much to prevent the establishment of this disease. They are as follows:

I. From earliest childhood, stand erect, walk erect, sit erect, never stoop, always let the weight of the shoulders fall behind you.

II. Keep your chest fully expanded by taking constantly, all your life long, full breaths so as to fully expand your chest. Do this at all times. Remember, you can not have consumption until your chest shrinks in size, either wholly or partially; so if you keep your chest flexible and constantly expanded, you will be safe from consumption.

III. Never let a cold run on you. Break it at once by taking active physic and cough medicines, and putting your feet at bedtime in hot water; keep them in until you get in a perspiration, and then go to bed and keep up the perspiration with hot drinks (Thompson's old "Composition Tea" is one of the best to use to start perspiration; hot lemonade is good, too); then take a portion of physic, and the next day your cold will be well. By pursuing this course for a length of time you get out of the habit of taking cold, and will rarely take one. Always continue your treatment until your cold is well.

IV. Avoid all debauching courses that weaken and reduce your constitution, such as soaking with liquor and actual drunkenness and dissipation of all kinds and gluttony and late night exposures. In fact, lead an honest, orderly life, free from vice and every dissipation; your health will then be equal, regular and constant, and your life, a long and happy one.

V. Keep your bowels always free by habit, diet, or purgatives.

Remarks—If these rules are strictly enforced by parents, with their children, when small, and by themselves, as soon as they can be made to understand their importance, very much will be done to improve the general

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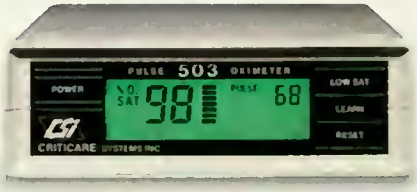

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health, as well as to prevent consumption. None are too old to take counsel from Rules IV. and V., and I might say also, from Rule III.

M.J. Guyot informs the profession that the phosphate of lime, in the rapidly exhausting night sweats of consumptives, is not only an almost specific cure, but tends also to improve the general health. ***Dose***—From 30 to 40 grs. in a little sweetened water, at night.

Dr. M.M. Griffith, of Bradford, Pa, claims that out of 25 cases of well-marked consumption, treated by small doses of crude petroleum, 20 are, to all means of diagnosis, cured; the rest have been materially benefitted, and none have been under treatment more than four months. The nausea attending the use of ordinary crude petroleum led him to adopt the semi-solid oil that forms on the tubing of wells. This, made into 3 to 5 gr. pills by incorporating an inert vegetable powder, was administered from 3 to 5 times a day in 1 pill doses. The first effect, he says, is the disappearance of the cough; night sweats are relieved, appetite improved, and weight is rapidly gained. These favorable symptoms continue until the patient is entirely recovered. If half of what Dr. Griffith claims shall prove true, generally, he has indeed made a valuable discovery. I hope, as *Scientific American* remarks, that Dr. Griffith has not mistaken some self-limiting phase of throat or bronchial disease for true consumption of the lungs; also, that continued trial of the alleged remedy will justify the high opinion he has formed in regard to its efficacy.

Dr. Talbot Jones, in a communication to the *New York Medical Journal* says there are 3,000,000 people who die annually of consumption; and also says "that climate is the physician's only dependence for the cure of his consumptive patient." Dr. Jones commends the climate of Minnesota for those predisposed to consumption, or laboring under its first stages, and thinks "that a residence there would be very likely to cure, or materially benefit them," and adds: "Between the pleasant rolling prairie, the wooded lake region, and the dense pine forests of the northern section of the state, they can choose what seems most agreeable and best adapted to them, while the dry, bracing atmosphere will enable them to live much of their time out of doors without fear of taking cold." He insists, however, as I have always done, that it is no use to send patients thither who are in the advanced stages of the disease. And this I know to be a fact.

The following item by E.R. Ellis, M.D., in the Detroit "News," in November, 1880, is so sensible and pertinent to the subject, I give it in full. He says: "Physicians are usually, and sometimes excusably, reluctant to advise invalids to go away from home and friends, and thus the matter is delayed, until a fatal result is inevitable. But every consumptive patient of mature years may know this for himself. If, in the favorable weather of summer and autumn, he is declining, with increased cough and shortness of breath, and occasional spitting of blood, his condition is alarming. He should change his physician, or climate, or both, immediately. If, with the above, his pulse is habitually up to or over 100 in a minute, a destructive process is going on, which, in this climate, the most skillful physician can arrest in not more than one case in four. In all such cases, go south at once, if not too far gone already. The quack here will encourage you to stay and make you brilliant promises up to the time of your death, but it is your own loss and folly if you believe him."

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Respiratory Physiology in Anesthetic Practice, by Thomas J Gal MD. Illustrated, 221 pages, hardcover. Baltimore: Williams & Wilkins, 1991. $42.00.

Dr Thomas J Gal, the eminent academic anesthesiologist from the University of Virginia, has developed a text that , as he proposes, in the Preface "is not intended to provide authoritarian information" nor "serve as an introductory text or handbook." One immediately wonders what else **Respiratory Physiology in Anesthetic Practice** might be. Upon a careful reading, I discovered a text that was short on encyclopedic minutiae but long on clinically relevant material, covering everything from the lung as an air pump to the use of artificial support devices. However, Dr Gal intends the book for a somewhat narrow audience, namely anesthesia providers, and readers outside this realm may be somewhat disappointed.

The book is broken down into six parts (namely, 4 major sections containing 11 chapters, an appendix, and an index). Chapter 1, "Basic Mechanics of the Respiratory System," is a brief (18 pages) treatment of the movement of air in and out of the lungs, a description of static and dynamic lung volumes, and a discussion of airway resistance and its measurement. This chapter is generally well written, concise, and nicely illustrated but unfortunately gets off to a bad start when it informs us that the dome of the diaphragm "ascends" to expand the thorax (Page 4), when in fact the diaphragm descends during inspiration in the nonparalyzed subject. This mistake is mitigated somewhat in that the correct relationship is demonstrated in the attendant figure. The closed-circuit helium-dilution and open-circuit nitrogen-washout techniques for lung volume estimation are mentioned but only in passing. The chapter is lavishly based in physics—the material requires it—but the text is well defined and the formulas are explained. Finally, Figure 1.9 (on Page 19) is amateurish in construction as it appears to have a "tilted" appearance on the page, and a couple of the labeling phrases have uneven baselines.

The next 20 pages of the book are devoted to Chapter 2, "The Physiology of Pulmonary Function Testing." The material is factual and succinct but uncreative or novel in presentation. The chapter's graphics borrow heavily from Gal's chapter in **Anesthesia** (a definitive American anesthesia text edited by Ronald Miller) as does much of the book; as one familiar with the Miller text, I find the current offering a somewhat watered-down version. Although such repetition is common in the medical textbook field, I find the use of recycled figures and graphs in new texts by the same author to be somewhat bothersome and suggestive that the author simply did not want to take the time to improve upon the original versions. Nonetheless, the sections on flow-volume loops and pulmonary function testing in surgical patients are well written and offer excellent clinical and interpretive information; unfortunately, the section on preoperative measures to improve lung function is too brief (3 paragraphs) and too poorly referenced (4 references, all before 1980) to be of any real value to the reader.

Chapter 3 is particularly specific to the technical and pharmacologic world of anesthesia practice and will have limited interest outside this arena. The influence of tracheal intubation and the major forms of anesthesia (regional block versus general) are discussed, and it is clear from the reading that this is where the author excels. The sections on muscle relaxants and tracheal intubation are nicely done and conceivably could provide readers outside of anesthesiology with some useful information. However, the discussion directed at the issues involving bronchospasm and its therapy is a regurgitation of previous work and is quite bland. Curiously, the chapter is somewhat over-referenced (107 citations) for a book that according to Dr Gal was intended "*not* to provide authoritarian information on a broad scale."

Section II ("Control of Breathing") consists of Chapters 4 ("Basic Respiratory Control"), 5 ("Control of Breathing in Normal and Disease States"), and 6 ("Effects of Drugs on Respiratory Control"). Here again, I am disappointed to find previously published figures (from Gal's chapter in **Thoracic Anesthesia** edited by Joel Kaplan) appearing with such frequency. The section is well written and brief, and the discussion of the measurement of ventilatory control mechanisms provides the reader with good information not only with respect to clinical management but also as a nice background for developing or interpreting clinical studies involving the ventilatory response. However, my general belief is that those individuals sophisticated in respiratory care will find little new or innovatively presented material.

Section III ("Blood Flow and Gas Exchange") contains two chapters, "Pulmonary Circulation" and "Respiratory Gas Exchange." Of particular interest to anesthesia-oriented readers is the discussion on hypoxic pulmonary vasoconstriction, which

includes treatment of clinical and pharmacologic modifications of this normal vasoconstrictive reflex. However, the section is unimaginatively illustrated, and the important portion entitled "causes of hypoxemia" is too brief and includes consideration of diffusion, shunt, and ventilation-perfusion etiologies. Later in the book, the fourth major cause of hypoxemia (hypoventilation) is introduced under the heading of "impaired gas exchange during general anesthesia." It is a shame that the author did not include some of the beautifully illustrative work that has been accomplished (primarily in Europe) over the last several years using computerized tomography mapping of positional and pharmacologically induced derangements in lung volumes.

The last section, entitled "Physiology of Mechanical Ventilation and Applied Airway Pressure," consists of three chapters, and is likely to be the most interesting to readers of RESPIRATORY CARE. This is the best part of the book in my estimation because Gal provides us with a wealth of clinically useful information in a highly rational, organized, and well-written manner. Chapter 9, "Basic Concepts of Ventilation," is largely a treatise on airway pressure and how the various phases and modes of ventilation (eg, inspiration, expiration, PEEP, and end-inspiratory pause) impact on this clinically useful composite of all the pressures transmitted to the airway during mechanical ventilation. The discussion of the cardiorespiratory effects of PEEP is brief but excellent, and broadens our understanding of why cardiac output falls when PEEP is applied (it is much more than just decreased venous return). The chapter is well illustrated in a manner that is both creative and utilitarian. Chapter 10 ("Monitoring Respiratory Mechanics during Mechanical Ventilation") and Chapter 11 ("Approaches to Mechanical Ventilation: Old and New") could have been combined, but even as separate entities they were thoughtfully conceived and are nicely presented. I especially enjoyed the concise presentation of inverse-ratio ventilation, airway pressure-release ventilation, and the various high-frequency modalities. Readers of the Journal may very well find these sections somewhat unsatisfying ("the exact mechanism for gas exchange under high-frequency oscillation is uncertain but has been the subject of excellent reviews," Page 195).

All in all, after I had spent 5 reading sessions spanning nearly 14 hours, 220 pages with 513 cited references were digested in relatively satisfactory terms; to me this indicates that the process was enlightening, without the pain of having to endure overwriting or amateurish writing. I still wonder what 'place' the book has (remember according to the author it is neither "authoritative" nor "a handbook" nor an "introductory text"). And while I tried to see the book in terms of what the author called a presentation "as a series of discussions," it just seemed like another "authoritative," albeit condensed, text. I give it a strong 'thumbs up' for the anesthesia crowd, and a somewhat less enthusiastic recommendation for those other readers of RESPIRATORY CARE.

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Clinical Blood Gases: Application and Noninvasive Alternatives, by William J Malley MS CPFT RRT, and edited by Eric D Bakow MA RRT, John S Capps BS RRT, Dean Hess MEd RRT, and John J Marini MD. Illustrated, 379 pages, hardcover. Philadelphia: WB Saunders Co, 1990. $36.00.

The author, William J Malley, has had 20 years of experience in teaching the concepts of clinical blood gases and, of equal importance, he has the clinical experience necessary for practical application of those concepts. According to the author, this book is designed primarily for the instructor and student who are charged with the responsibility of teaching and learning clinical blood gas information. At $36.00, it can be a valuable addition to the library of the respiratory care educator, and is equally suited for the clinician's use in reinforcing clinical blood gas knowledge and as a reference for hospital-based clinical education.

Chapter 1 introduces blood gases beginning at a basic level. I agree with the author that the reader need not have had previous health care experience; clearly, however, the reader must be familiar with scientific language. The author does an excellent job of presenting a complex subject in a fashion that does not intimidate the reader, while at the same time causing the reader to understand the importance of this procedure. Figures 1-3 through 1-10 are especially helpful to the reader for defining anatomical landmarks associated with arterial blood.

Chapters 2 and 3 build logically upon the first chapter by describing common errors associated with evaluation of arterial blood gas (ABG) data. The rather complex issues of electrode function and ABG quality control are presented simply and are therefore not likely to intimidate the student. The respiratory care instructor will find the manner of presentation of this material conducive to good learning.

Chapters 4 thru 14 walk the reader through basic physiology associated with the human body's ability to oxy-

genate, the interpretation of basic acid-base values, and the assessment and treatment of various clinical conditions.

Chapters 15 and 16 describe various noninvasive monitoring techniques and provide relevant case studies. The value and the manner of presentation of Chapter 15 are excellent. Noninvasive blood gas monitoring is a subject about which great misunderstanding exists in clinical practice, caused primarily by the proliferation of technology. This chapter provides an excellent opportunity for the reader to better understand the clinical issues and thus to decide how to best utilize this technology in clinical practice. Chapter 16 provides the reader with an opportunity to apply the knowledge presented through the previous 15 chapters to detailed case studies. The breadth of cases provided, coupled with questions specific to each case, challenge the learner to better understand the clinical utility of data provided.

The number and quality of illustrations, tables, and graphs found throughout this book support learning. Exercises following each chapter are especially helpful in reinforcing material presented. I believe that students and instructors alike will benefit from this systematic approach, which provides an excellent basis for understanding clinical blood gases. This book will likely be a frequently consulted reference long after graduation. Because the author relates the basic sciences to the clinical application of blood gases, this book is a welcome addition to the library of any allied health educator or respiratory care department.

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Diffuse Diseases of the Lung: A Team Approach, by WM Thurlbeck MB, Roberta R Miller MD, Nestor L Müller MD, Edward C Rosenow III MD. Illustrated, 243 pages, hardcover. Philadelphia: BC Decker, 1991. $79.00.

With esteemed authors representing expertise in pathology, radiology, and clinical pulmonary disease, **Diffuse Diseases of the Lung: A Team Approach** presents an integrated approach to pulmonary disease. The book consists of 13 chapters with 236 pages of text. The first 3 chapters deal with normal lung structure and function, abnormalities of structure and function, and practical considerations in obtaining lung biopsies. As a result of the broad-reaching expertise of the authors, discussions of structure and function correlate physiology, pathology, and imaging studies extremely well. Chapters 4 thru 13 are devoted to specific groups of pulmonary disorders—eg, diffuse pulmonary hemorrhage (Chapter 6), collagen vascular disease (Chapter 8), chronic airflow obstruction (Chapter 12), and pulmonary vascular disease (Chapter 13).

The strength of this book lies in the integrated approach that stems from the marriage of different chest disciplines among its authors. However, with the major focus on diagnostic recognition and patterns of disease, treatment is beyond the scope of the book and is not discussed.

The book is generously illustrated with photographs of chest-imaging studies (including x-rays and computerized tomographic scans), gross pathology specimens (light microscopy specimens), and electron microscopic sections. Also, summary tables are abundant to provide the appropriate clinical context.

Overall, **Diffuse Diseases of the Lung: A Team Approach** is not only especially valuable to pulmonologists and to advanced trainees, but also is a useful addition to respiratory care libraries. Respiratory care practitioners seeking a detailed diagnostic description of some unusual pulmonary entities will find this a valuable reference text.

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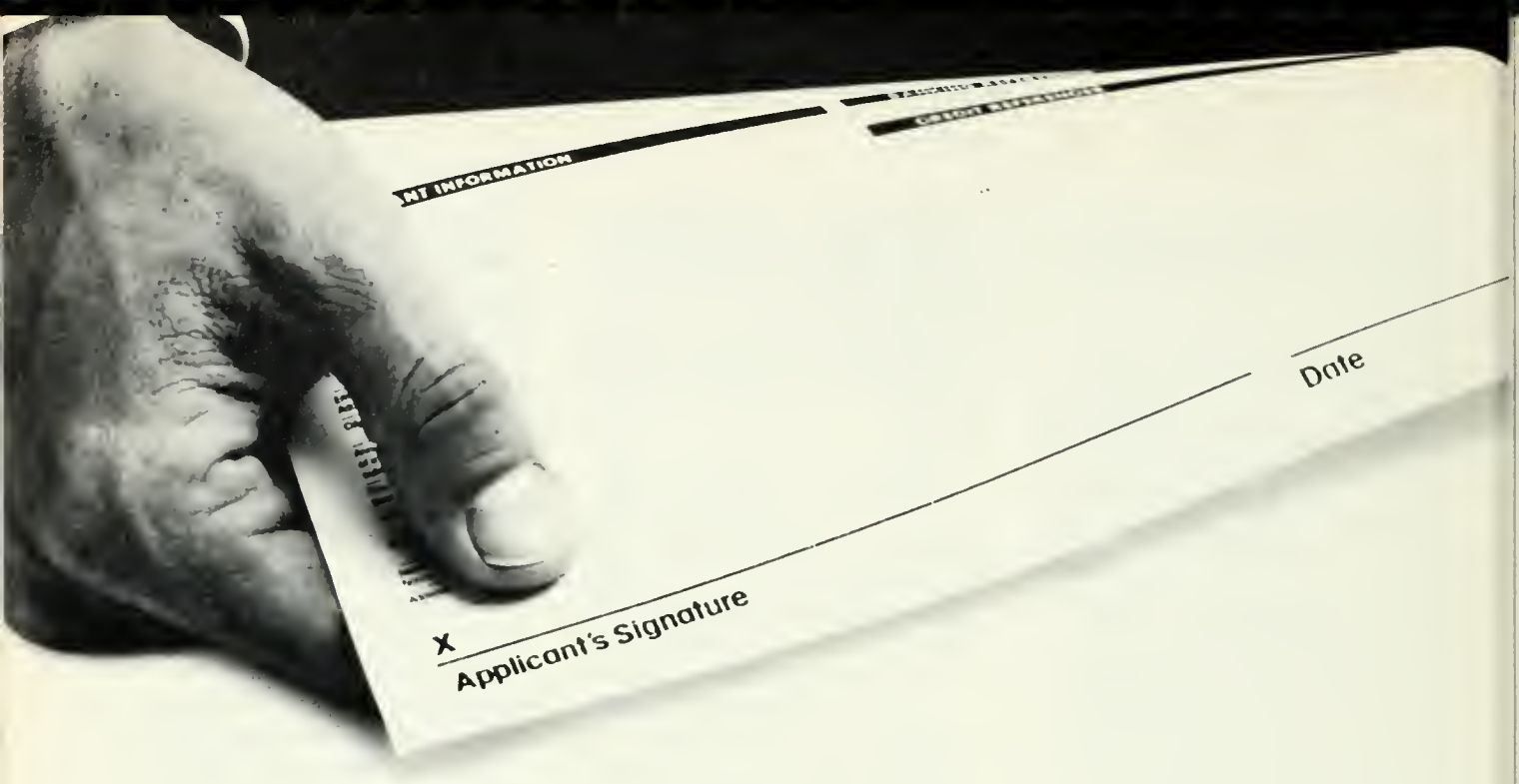
The Oxygen Status of Arterial Blood, edited by Rolf Zander and Friedrich Mertzlufft. Illustrated, 294 pages. Munich, Germany: S Karger GmbH, 1991. $63.25

This delightful little book is a "must read" for anyone interested in the technical details surrounding the measurement of oxygen in blood, a well-edited summary of a conference of clinicians and physiologists held in Mainz in May 1990. The stated goal of the conference was to "surmount the often criticized tendency for theory and practice to drift apart." In this volume the editors have succeeded well. Of the 20 contributors, all but one is from the Federal Republic of Germany.

Originally published in German, this book is remarkably readable, due in part to the translation assistance of Dr Martin Reddington. An occasional Latin phrase for a common condition is used, but this adds to the interest rather than detracting from the message. Beginning students will find the vocabulary difficult and I don't recommend this book for those naive about blood gas terminology. An occasional European spelling occurs, such as "centre" for "center," but overall the translation is excellent. The antiquated terms "blue bloater" and "pink puffer" are used for these forms of chronic lung disease. "Blockage of the sympathet-



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icus" is used for "sympathetic blockade," "artificial respiration" for "mechanical ventilation," and "pulmonalis catheter" for "pulmonary artery catheter." Thankfully, both SI and conventional units are presented in the text.

The book consists of 32 separate papers (chapters) organized around the components of the oxygen delivery system and in five major sections: Arterial O_2 Partial Pressure, Arterial O_2 Saturation, Concentrations of Hemoglobin and Hemoglobin-Derivatives, Arterial O_2 Concentration, and the O_2 Status of Arterial and Mixed Venous Blood. A brief, one-paragraph summary follows each paper. Consensus opinions are summarized at the end of each section.

The 9 papers in the first section include short papers on differential diagnosis of arterial hypoxia, limitations of analytical methods and analyzers, invasive and noninvasive, and intermittent and continuous monitoring of arterial O_2 partial pressure. A thorough discussion of the methods and limitations of each technique is presented. Clinical data are summarized and consensus recommendations are listed.

The next section, Arterial O_2 Saturation, consists of 7 papers, including the causes of low arterial saturation, in-vitro and in-vivo techniques of measurement, quality control and calibration concerns, continuous invasive and noninvasive techniques, and a very interesting concluding paper "Normal and Therapeutic Threshold Values for Arterial O_2 Saturation." In this latter paper a theoretical discussion of the clinical low-limits (safety) of O_2 saturation is presented; a similar discussion is included in each of the other sections that, although somewhat repetitious, contributes greatly to an understanding of the differences of each measure of adequacy of arterial oxygenation.

The next eight papers deal with the hemoglobin component of the oxygenation system, with measurement and effects of the various species of hemoglobin on different monitoring systems addressed. Contributions of smoking and the resultant effects of carbon monoxide on the shape of the dissociation curve and measurement of arterial O_2 saturation are considered separately. Two interesting and unique papers are Therapeutic Thresholds for Acute (1) and Chronic (2) Alterations in Hemoglobin Concentration. An extended discussion about "optimal" hematocrit is provided, although the supporting references are minimal. In these papers, the dangers of increased hemoglobin (> 17 mg%) are raised, a topic rarely addressed in contemporary American texts.

The next section, "Arterial O_2 Concentration," consists of seven papers beginning with the causes of arterial hypoxemia. It is an abbreviated discussion of all possible alterations in gas exchange, which is a repeat of previous discussions and very superficial. Methods of measurement are discussed including several not in use in this country, including fuel-cell and photometric technology applied to arterial blood.

The last two papers form the section on O_2 status of arterial and mixed venous blood. The first of these is the longest in the book and deals with the theoretical issues of mixed venous O_2 ($P_{\bar{v}O_2}$). It provides a good brief review of the utility and theoretical basis for this monitor; the multiple factors influencing $P_{\bar{v}O_2}$ are discussed in detail, including the effects of cardiac output.

All of the papers are succinct, usually 4-8 pages. They contain a limited reference list (4-10 references) that is somewhat dated (the most recent reference was 1989, most are before 1980) and almost entirely from the German literature. This severely reduces the value of this book to American audiences. The strengths of the monograph are its consistent editorial style, brief overviews, and consensus opinions; however, the consensus opinions are mostly unsupported by accessible references.The sections on equipment and measurement techniques are very valuable. There are a few important but uncommon expressions used in this book; fortunately, these are well described in an extended and detailed appendix. For instance, "hypoxygenation" is used to mean decreased O_2 saturation, while "hypoxic hypoxygenation" is hypoxemia as a result of decreased P_{aO_2}. "Toxic hypoxygenation" is reduced saturation resulting from abnormal hemoglobin binding of oxygen (such as due to methemoglobinemia or carbon monoxide). These and other terms are at first confusing, but they are used consistently and frequently throughout the book.

Overall this text's value is in its consensus statements (with the limits noted above) reflecting the European (German) expert opinion. These should be stimulating to American readers and be used to challenge the validity of our own deeply held prejudices. However, the primarily German references limit the usefulness of this text as a resource volume.

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Calendar of Events

AARC & AFFILIATES

April 13-15 in Philadelphia, Pennsylvania. The PSRC presents its 27th Annual Conference and Exhibition, "Foundations and Discoveries," at the Adam's Mark Hotel. Contact Betsy Schneck (215) 829-3578 or Kathy Yandle (215) 453-4517.

April 14-16 in Fargo, North Dakota. The NDSRC presents its Annual Respiratory Care Convention. "Current and Future Spirit of Excellence," at the Doublewood Inn. Lecture topics cover legislative activities, monitoring systems, and patient care trends. Contact Kathleen Hanson, Dakota Hospital, 1720 University Dr Fargo ND 58103. (701) 280-4100.

April 15-17 in Des Moines, Iowa. The Iowa Society for Respiratory Care presents the 14th Annual Iowa Lung Conference at the Des Moines Marriott Hotel. Topics include interpersonal relations in the work place, preparation for the CRTT and RRT exams, CLIA update, job interviewing, non-traditional bronchodilator delivery, mechanical ventilation, and nutritional support. Special speaker is Bob Gibson, baseball hall-of-famer. Contact Mike Wheeler RRT, Respiratory Care, Iowa Lutheran Hospital, Des Moines IA 50316. (515) 263-5108.

April 16-17 in Lawrence, Kansas. The KSRC presents its 15th annual education seminar. Topics include pulmonary rehab, professionalism, the resurgence of TB, and asthma. A golf tournament and Sputum Bowl are scheduled for the 15th. Contact Frank Hart, Pulmonary Services, Shawnee Mission Medical Center, 9100 West 74th St, Shawnee Mission KS 66201. (913) 676-2175.

April 21-24 in Cincinnati, Ohio. Ohio, Kentucky, and Indiana present the 19th Annual Region II for Respiratory Care meeting at the Albert B Sabin Convention Center. The event begins April 21 with four concurrent specialty programs: alternate site ventilation, people skills, lab quality assurance, and laughter in nicotine addiction. The regular meeting then continues April 22-24. Other events include over 100 vendor exhibits and Sputum Bowl competitions. Contact Michael Moore (513) 221-3050.

April 22-24 in Sioux Falls, South Dakota. The SDSRC presents its Annual Meeting at the Howard Johnson. The program features workshops on equipment, hemodynamic monitoring, weaning the unweanable, updates on cystic fibrosis, and health care reform. Speakers include Senator Tom Daschel, Dr David J Pierson, and AARC Vice President Tommy Rust. For information, contact Liz Gray at (605) 333-6302.

April 29-30 in Waterbury, Connecticut. The CSRC presents the 11th Annual Super Symposium at the Sheraton Hotel. Topics include ultra high frequency jet ventilation, ECMO, pressure control ventilation, and rehab. Social events include Wallyball and a dance. A golf tournament is planned for the 28th. Contact Dan Madison, CSRC, PO Box 3256, Enfield CT 06083. (203) 524-3212.

April 29-May 1 in Osage Beach, Missouri. The MSRC presents its 21st Annual Educational Seminar at the Tan-Tar-A Marriott. Topics cover a wide range of subjects, including care of the caregiver, CQI, and respiratory protocols. Contact Wilma Alexander (816) 276-4222.

May 3-5 in Bellevue, Washington. The 19th Annual Pacific Northwest Regional Respiratory Care Conference is slated for at the Bellevue Red Lion Inn. Topics cover pediatric, management, and critical care issues. A special guest speaker addresses the establishment of a 4-year respiratory care program in Washington State. Call Richard Larson (206) 880-4585.

May 6 - AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents "Weaning from Mechanical Ventilation," one in a series of live satellite videoconferences titled "Professor's Rounds in Respiratory Care." Featured presenters are Drs. Martin J Tobin and David J Pierson. Site registration for entire staff is $245 for AARC members. Call (214) 830-0061.

May 6-8 in Madison, Wisconsin. The WSRC presents its 22nd Annual Convention at the Holiday Inn-West Hotel and Convention Center. National and local speakers present lectures for practitioners of all specialties. Call Tami Meier, RRT, at (608) 263-7050.

May 7-9 in Los Angeles, California. The CSRC Chapter IV presents the 2nd Neonatal/Pediatric Pulmonary Care Conference and Golf Tournament. Topics include PFTs, HFV, asthma, synchronous infant ventilation, PEP & autodrainage therapy, cardiac surgery update, and surfactant controversies. The cost is $45/day with

lunch included. Contact Kelvin MacDonald, Kaiser Permanente, (213) 667-6635.

May 13-15 in El Paso, Texas. The Southwest Region, of the TSRC presents its 21st Annual Conference, "Updates '92," at the Westin Paso Del Norte Hotel. Contact Yvonne Banegas, Respiratory Care, Providence Memorial Hospital, 2001 N Oregon, El Paso TX 79902, (915) 542-6563.

May 27-29 in Myrtle Beach, South Carolina. The Georgia/South Carolina Region VI Committee presents its Annual Conference and Assembly at the Landmark Hotel. Topics include the chemically impaired practitioner, PEP mask therapy and care of the elderly. Contact Bob Barber, Spartanburg Regional Medical Center, 101 E Wood St, Spartanburg SC 29303, (803) 591-6524.

May 28-29 in Laurel, Maryland. The MD/DC Society for Respiratory Care holds its 2nd Annual "Conference by the Tee" at Patuxent Greens Country Club. Topics include PC/IRV, ACLS and the RCP, CLIA validation protocols, continuous-flow ventilation, and trends in mechanical ventilation. Contact Joe Lynott (202) 877-1064.

June 3-5 in St Charles, Illinois. The ISRC presents its 23rd Annual State Convention at Pheasant Run Resort. This year's theme, "Looking Back to Your Future," takes a retrospective and future look at respiratory care. The meeting features lectures, an entry-level review workshop, management workshop, exhibits, entertainment, and a special minisymposium titled "Future Challenges for Health Care Providers." Contact Doug McQuerary (312) 962-4060 or Jane Reynolds (312) 883-6535.

June 3-5 in Checotah, Oklahoma. The OSRC presents its 27th Annual Seminar, at the Fountainhead Resort on Lake Eufala. The program features lectures and exhibits. Contact Betty Fisher (405) 636-7065.

June 10-12 in Roundtop, New York. The New York and New Jersey Societies jointly hold their 5th Annual Managers and Educators Conference at the Winter Clove Inn. For information, contact Patty Bowe (519) 270-7454 or Ken Wyka (201) 456-7228.

OTHER MEETINGS

April 9-10 in San Diego, California. The California Society for Pulmonary Rehabilitation (CSPR) presents the 3rd annual state meeting "Charting New Horizons in Pulmonary Rehabilitation" at the U.S. Grant Hotel in San Diego. Contact Mary Jo Zanella-Goldzimer, program chairperson, 3335 Country Rose Circle, Olivehain CA 92024, (619) 740-4584 (beeper).

April 27-28 in Virginia Beach, Virginia. The California College for Health Sciences (CCHS) presents "The 90's Staffing Solutions in Respiratory Care" seminar at The Cavalier Hotel. Contact Doria Pasqual, CCHS, 222 West 24th St, National City CA 91950, (619) 477-4800.

May 3-5 in Bellevue, Washington. The 19th Annual Pacific Northwest Regional Respiratory Care Conference will be held at the Bellevue Red Lion Inn. Topics cover pediatric, management, and critical care issues. Also, a guest speaker will address the establishment of a four-year respiratory care program in Washington state. For details, call Richard Larson at (206) 880-4585.

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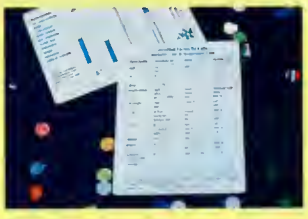

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2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

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3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139s-143s.

Abstract in journal: (Abstracts are not strong references. Abstracts more than 3 years old should not be cited. When cited, abstracts should be identified as such.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35:1087-1088.

Editorial in journal:

6. Rochester DF. Does respiratory muscle rest relieve fatigue or incipient fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Editorial with no author given:

7. High frequency ventilation (editorial). Lancet 1991; 1:706-708.

Letter in journal:

8. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (Specific pages should be cited whenever possible.)

10. Nunn JF. Applied respiratory physiology. New York: Appleton-Century Crofts, 1969.

Note: To specify pages in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1969:85-95 (series of contiguous pages). 1969:85,95 (separated pages).

Corporate author book: (Specific pages should be cited whenever possible.)

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with editor(s): (Specific pages should be cited whenever possible.)

12. Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in book: (Specific pages should be cited whenever possible.)

13. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Phildelphia: JB Lippincott, 1977:171-223.

Newspaper article:

14. Rensberger B, Specter B. CFCs may be destroyed by natural process. The Washington Post 1988 Aug 7:Sect A:2(Col 5).

Dictionary or similar reference:

15. Pneumohemopericardium. Dorland's illustrated medical dictionary, 26th ed. Philadelphia: WB Saunders, 1981: 1038.

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1992 Call for Abstracts

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in San Antonio, Texas, December 12-15, 1992. Accepted abstracts will be published in the November 1992 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications—READ CAREFULLY!

An abstract may report (1) an **original study,** (2) the **evaluation of a method or device,** or (3) a **case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

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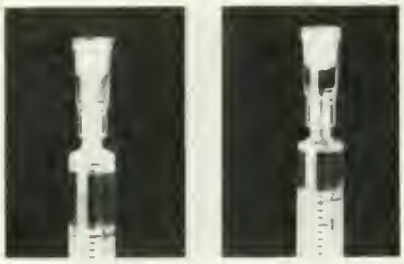

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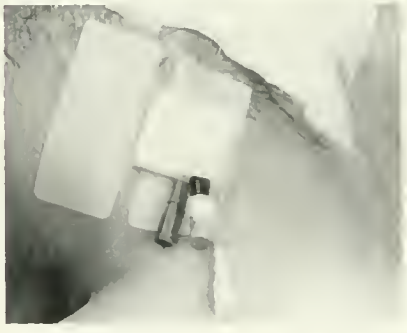

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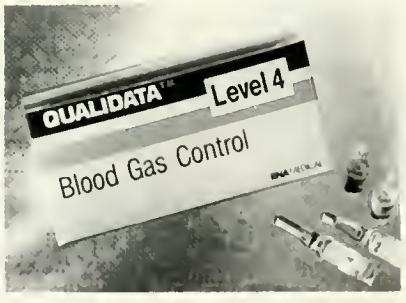
PEAK FLOW METER FOR HOME MONITORING. Wright Pocket Flow Meters for home monitoring, standard range model (80-780 L/min) and low-range model (50-400 L/min), meet the NAEP Technical Standards for peak flow meters and offer superior durability, ease of use, a 5-year warranty, and a 5-year useful life, according to the manufacturer. Ferraris Medical Inc, Dept RC, 9681 Wagner Rd, PO Box 344, Holland NY 14080, (800) 724-7929.



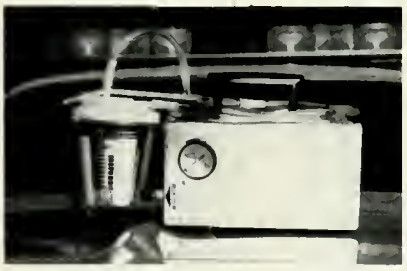
BLOOD GAS/ELECTROLYTE CONTROL WITH ELEVATED P_{O_2} VALUES. In addition to normal control values for pH, P_{O_2}, Na^+, K^+, Cl^-, and Ca^{++}, RNA's new Blood Gas/Electrolyte Control also offers elevated P_{O_2} values in the 250-315 torr range that is useful for blood gas labs that monitor patient specimens with artifically elevated P_{O_2} levels. This control comes in 2-mL ampules, and has a 3-year shelf life at room temperature. Users are eligible to participate in RNA's monthly statistical service that helps labs document accuracy and precision. RNA Medical, Dept RC, 2 Craig Rd, Acton MA 01720, (508) 263-6162 or outside MA (800) 533-6162.

CATALOG. 1992/93 catalog with up-to-date information on over 50,000 products from more than 400 leading suppliers serving clinical and industrial laboratories. Color tabs and indexes (main and mini) direct you to your chosen section, according to the manufacturer. For your personal copy, direct a request on your hospital or company letterhead to Curtin Mathewson Scientific Co, Dept RC, Marketing Dept, PO Box 12546, Houston TX 77251.

REUSABLE FILTERS. According to the manufacturer, a new family of HEPA-rated (0.3 μ, 99.97%), rugged, high-quality filters reduces costs and cuts medical waste, getting better retention with each autoclaving. Mainline respiratory filters can last for up to 1 year and are environ-

mentally responsible. Reusable/autoclavable filters for inspiratory lines could significantly reduce filter costs, with a bright green housing to identify them as reusables. Arbor Medical (313) 665-3300. Mention RESPIRATORY CARE when you call.

NASAL CPAP SYSTEM. REMstar Choice Nasal CPAP System offers wireless remote control with convenient push-button operation that enables patients to choose the most convenient location for their unit, up to 12 feet away from the bed. Designed to enhance the patient's comfort as he or she falls asleep, this system begins delivering air at a pressure of 2.5-3 cm H_2O, and over a prescribed, preset time period, the air pressure gradually increases until full prescription pressure is reached. Respironics Inc, Dept RC, 1001 Murry Ridge Dr, Murrysville PA 15668-8550. (412) 733-0200.



BATTERY-POWERED SUCTION UNIT FOR HOSPITAL USE. S-SCORT Jr (#90012A) provides the value of economy in a hospital-grade suction unit, delivering 550 torr negative pressure and 32 L/min airflow to the end of the patient tubing. According to the manufacturer, the unit exceeds AHA standards for CPR, utilizes disposable collection components, and is available from most hospital supply distributors for safe and economical clean-up and disposal. The unit utilizes sealed lead-acid batteries and fixed-volt chargers. SSCOR Inc, Dept RC, 11064 Randall St, Sun Valley CA 91352. (818) 504-4054.

PFT/BODY PLETHYSMOGRAPH SYSTEM. According to the manufacturer, MasterLab PFT system is easy to use and lets the user choose the sequence and content of each patient report, storing more than 100 custom report formats. Tests include complete flow-volume loops, slow VC, MVV, single-breath D_{LCO}, helium FRC, airway resistance, and TGV. According to the manufacturer, accurate and reliable measurements are made with normal tidal breathing; so, the patient does not have to perform stressful panting. Sterilization is fast and easy because MasterLab uses a unique valve block that is effortlessly removed for cleaning. Quinton, Dept RC, 2121 Terry Ave, Seattle WA 98121. (800) 426-0337.

CO-OXIMETRY CONTROL FOR CORNING 2500. Alko Diagnostic Corp has developed and introduced a dye-based CO-oximeter control for the Ciba-Corning 2500 CO-oximeter that comes in three levels. According to the manufacturer, this control is free of all biohazards, does not require special handling or refrigeration, and simulates the absorption values of whole human blood that are physiologically correct. A free, dependable monthly statistical program is available. Alko Diagnostic Corp, Alko Park, Dept RC, 333 Fiske St, Holliston MA 01746. (508) 429-4600.

LIQUID OXYGEN SYSTEMS. The liquid-oxygen breathing systems of John Bunn Co and Inspiron Corp were recently acquired by Andonian Cryogenics Inc; product line has been expanded to include both new and factory-rebuilt reservoirs with 30-L and 58-L capacities, portable units with a 1.2-L capacity, and a compact, lightweight 1/4-L "walk-around" unit. As a further convenience to dealers, the liquid oxygen systems are also available on a rental basis. Andonian Cryogenics Inc, Dept RC, 26 Farwell St, Newtonville MA 02160. (800) 446-3533 or (617) 969-8010.

REMEMBER!

OPEN FORUM Abstract deadline is June 6, 1992

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– The result of this development is a safe, user-friendly, high efficiency unit that puts the technology where it counts — at your fingertips. Over the last 10 years, clinicians have been converting to heated wire circuits to reduce humidity rainout. Now, with new requirements to red bag condensate from watertraps and circuits, the need to reduce or eliminate rainout is increasingly important.

SAFETY

The DSC 17000 design places the humidifier chamber beside the electronics so that spills onto the chamber will drain around the chamber base through drain holes, protecting the unit from damage.

The DSC 17000 heated wire design keeps patient safety in mind by never allowing the chamber air temperature to exceed the distal patient temperature setting (heated wire mode only). When changes in flow occur, high temperature gas will not be delivered to the patient's trachea.

The DSC 17000 can provide a closed system without watertraps, reducing the need to open the circuit and reducing potential contamination. This set-up method provides a safer system for the patient.

CLINICAL DATA

Independent testing has shown the DSC 17000, chamber and circuits perform to high standards of safety and reliability. We have set our standards high, achieving faster warm-up time and more reliable temperature monitoring (even on long circuits with dual heater wires).

PRODUCT AVAILABILITY

The DSC 17000 is designed for use with adult, pediatric and neonatal patients.

Humidification Chambers:

- Adult low and high flow.
- Neonatal/pediatric, low and high flow, with low compressible volume.

Anesthesia and ventilator circuits

Temperature probes: 40^{M}; 60^{M}; 72^{M}

Mounting brackets:

- Bird
- Puritan Bennett
- Bear
- Siemens
- Pole Clamp.

For more information please contact your LDS representative.

LDS Life Design Systems A Division of Bird Medical Technologies Inc. BIRD
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New

2500 VOLDYNE®

Volumetric Incentive Deep-Breathing Exerciser

The accuracy of Voldyne, in a new size, matched to geriatric patients and patients with smaller lung capacities.

Voldyne 2500 ...

- A smaller, lighter flow cup **reduces the work of breathing**, thus improving patient performance and progress.
- Every unit is **individually tested and calibrated** for performance, reliability and superior accuracy of inhaled lung volume.
- Volume incentive spirometry **improves assessment of patient progress** by eliminating the guesswork associated with spirometers that only measure flow.
- Graduations printed on both sides of the unit allow the therapist to conveniently **observe volumes while instructing and encouraging the patient.**

For further information, contact your Sherwood O.R. / Critical Care Representative or call.

1-800-325-7472 (outside Missouri)

1-800-392-7318 (in Missouri)

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